# LOCATION OF PLACENTA ON ULTRASOUND AS A PREDICTOR TEST FOR HYPERTENSIVE DISEASE OF PREGNANCY

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**IN**

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**LUDHIANA**
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Dedicated

To

My

Parents

And

Teachers

# ABBREVATIONS

| | |
|---|---|
| AT | Angiotensin |
| APGAR | Appearance, Pulse, Grimace, Activity, Respiration |
| AST | Aspartate aminotransferase |
| ALT | Alanine Aminotransferase |
| ANOVA | Analysis of Variance |
| B2- | Beta 2 |
| BMI | Body Mass Index |
| BP | Blood pressure |
| Ca2+ | Calcium |
| CD | Cluster of Differentiation |
| DC | Decorin |
| eNOS | Endothelial nitric oxide synthase |
| ET1 | Endothelin |
| EVT | Extravillous trophoblast |
| EP1,2 | Prostaglandin receptors |
| Fox | Foxhead box |
| GFR | Glomerular Filtration Rate |
| HDP | Hypertensive Disease of Pregnancy |
| HcG | Human chorionic gonadotropin |
| HELLP | Hemolysis elevated liver enzymes, low platelet count |
| HLA | Human leukocyte Antigen |
| HLA DR | Human Leukocyte Antigen |
| IL | Interleukin |
| IUFD | Intrauterine fetal deaths |
| IUGR | Intrauterine Growth Restriction |
| IgM | Immunoglobulin |
| LDH | Lactate Dehydrogenase |
| LBW | Low birth weight |
| LFT | Liver Function Test |
| LBW | Low birth weight |
| mRNA | Messenger RNA |
| MHC | Major Histocompatability Complex |
| MAPK | Mitogen Activated Protein Kinase |
| NK | Natural Killer |
| NO | Nitric oxide |
| NADPH | Nicotinamide Adenine Dinucleotide phosphate hydrogen |
| NICU Admission | Neonatal Intensive Care Unit |
| PTD | Preterm delivery |

| | |
|---|---|
| PlGF | Placental Growth Factor |
| PIH | Pregnancy Induced Hypertension |
| P value | Probability value |
| PGI2 | Prostacycline |
| PGE2 | Prostaglandin E2 |
| PlGF | placental growth factor |
| RFT | Renal Function Test |
| RAS | Renin angiotensin System |
| SD | Standard deviation |
| SPSS | Statistical Package for the Social Science. |
| sFlt | Soluble fms like tyrosine kinase |
| Spot P:C Ratio | Spot Protein:Creatine Ratio |
| STBM's | Syncytiotrophoblast microvsicles |
| T-Lymphocyte | Thymus lymphocyte |
| Treg | Regulatory T cells |
| TxA2 | Thromboxane A2 |
| USG | Ultrasound |
| VCAM | Vascular Cell adhesion molecule |
| VEGF | Vascular endothelial growth factor |
| WHO | World Health Organisation |

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# ABSTRACT

**Background-**Hypertensive disease of pregnancy is the most common complication of pregnancy and is a major cause of maternal and perinatal morbidity. Hypertensive disorder of pregnancy, also known as maternal hypertensive disorder, is a group of diseases that includes preeclampsia, eclampsia, gestational hypertension and chronic hypertension. The placental location between 18-24 weeks on ultrasound can be used to predict the development of hypertension as hypertensive disease of pregnancy occurs as a disease of trophoblastic tissue. Placental abnormality is one of the initial events which lead to the pregnancy induced hypertension and it's detection on ultrasound can be used as a reliable predictor of hypertensive disease of pregnancy, that can be used to decrease the complication rate, mortality and morbidity occurring due to hypertensive disease of pregnancy.

**Aims and objectives-**To study correlation of placental on ultrasound as a predictor test for development of hypertensive disease of pregnancy.

**Material and Methods-** This study was a prospective study conducted in the department of Obstetrics and gynaecology, Dayanand Medical College and hospital, Ludhiana. The study was conducted on 100 women on outdoor as well as indoor women at 18-24 weeks of gestation, placental location was determined on ultrasound and were followed for the development of hypertensive disease of pregnancy.

**Result-**Out of 100 women, the women who developed hypertension with lateral placenta was 55% (n=22) and with central placenta was 33.3% (n=20). The abnormal outcomes like preterm birth (40%), low birth weight babies (68.2%), low mean APGAR score (5.73), NICU admissions (70%) were more in group with lateral placenta.

**Conclusion:-**This study showed significant association between lateral location of placenta and development of hypertensive disease of pregnancy. Location of placenta on ultrasound at 18-24 weeks can be used as a reliable, non-invasive, cost-effective predictive screening test for development of hypertensive disease of pregnancy.

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# INTRODUCTION

Pregnancy is one of the most cherished events in a woman's life. If there is any complication during pregnancy it may affect her mental and physical health. It is always good for the obstetrician to detect any high risk factor so that it can be rectified or managed in time.

Hypertensive disease of pregnancy (hypertensive disorder of pregnancy) is a group of diseases which includes preeclampsia, eclampsia, gestational hypertension and chronic hypertension.

Hypertensive disorder is one of the most common medical problem complicating pregnancy. It complicates up-to 7-10% of pregnancies of which Preeclampsia/Eclampsia contributes 70% and chronic hypertension 30%[1]. Pre-eclampsia, eclampsia contribute to the death of a woman, every three minutes[2]. This has led to the interest in screening and detecting this disorder. The most important factor is lack of timely prediction[3].

Preeclampsia is a complex clinical syndrome involving multiple organ systems and remains one of the major cause of maternal and perinatal morbidity and mortality[1]. Women who have history of preeclampsia in previous pregnancy are at a higher risk of development of preeclampsia in the subsequent pregnancies.

Preeclampsia is defined as persistent systolic blood pressure level of 140mmHg or higher and diastolic blood pressure of 90 mmHg or higher that occurs after 20 weeks of gestation with proteinuria in women with previously normal blood pressure[4,5].

The incidence of preeclampsia has increased by 25% during the past 2 decades[6]. Preeclampsia is one of the leading cause of maternal and perinatal morbidity and mortality with an estimated 50,000 -60,000 deaths per year worldwide[7,8]. For each preeclampsia related death that occurs, there are about 50-100 other women who experience ''near miss''. Near miss is the significant maternal morbidity that results in significant health risk and health care cost although does not result in death[9,10]. The infants

born to these mothers are small and premature, may experience prolonged stay in neonatal intensive care units and often face developmental delays.

Maternal Complications:-

Outcome of pregnancy which is complicated by hypertension ranges from uneventful pregnancy in women with chronic and controlled hypertension to death in cases of preeclampsia-eclampsia. The major adverse outcomes include central nervous system (CNS) injuries such as seizures, hemorrhagic and ischaemic stroke, hepatic damage ranging from transaminase elevation, HELLP syndrome (hemolysis, elevated liver enzymes and low platelets), hepatic failure, renal dysfunction (from trivial reduction in glomerular filtration to reversible acute renal failure or acute tubular necrosis to even irreversible renal failure which occurs secondary to the renal cortical necrosis).There is also increased risk for women with preeclampsia to develop end stage renal disease though the absolute risk appears to be low. There is also increased frequency of cesarean delivery, preterm delivery and abruption placentae[11-13] and it's related complications.

Fetal Complications:-

Preeclampsia-eclampsia can lead to increased incidence of induced labour, fetal growth restriction, neonatal respiratory difficulties and increased frequencies of admission to neonatal intensive care unit.. The effect of chronic controlled hypertension in pregnancy on fetus are minimal[11-13].

Numerous clinical, biochemical and biophysical tests in early or mid-pregnancy have been proposed for prediction of preeclampsia. The predictive tests used are related to placental perfusion and vascular resistance (mean second trimester BP, cold pressor test , isometric hand grip exercise, roll over test, intravenous infusion of angiotensin II, 24 hour ambulatory BP monitoring), fetoplacental unit endocrinology (alpha-fetoprotein, human chorionic gonadotropin), renal function (serum uric acid or microalbuminuria), endothelial dysfunction (antiphospholipid antibodies or homocysteine) and angiogenic factors which are circulating. Most of the

tests in this category are cumbersome, time consuming and are overall inaccurate.

There is abnormal trophoblastic invasion with vascular remodelling and consequent increased resistance in the spiral arteries in preeclampsia. These changes predispose to hypoxia-reperfusion injury in the placenta that leads to generation of excess load of free radicals and reactive oxygen species creating an alteration between angiogenic and antiangiogenic factors, vasodilators and vasoconstrictors[14].

The overall risk of preeclampsia in laterally located placenta is greatly increased due to inadequate development of blood supply. Placenta plays one of the major role in the pathogenesis of preeclampsia. It is found that the blood supply of placenta is not uniformly distributed. The site of placental implantation and thus the resultant location of placenta with in the uterus are likely important determinants of placental blood flow and the pregnancy success rate.

The placenta is known as central when it is present equally between right and left part of uterus irrespective of anterior, posterior, fundal location. When 75% or more of placental mass is located to one side of midline, it is known as unilateral right or left sided placenta[15].

The screening test which is choosen, ideally should be simple, inexpensive and reproducible with high sensitivity and positive predictive value. To ensure maximum patient acceptability, it should be easy to perform and non-invasive.

Prenatal care, earlier detection, careful monitoring and adequate treatment during pregnancy are essential to prevent the serious complications. It is important to identify women with preexisting hypertensive disorders, a prior history of hypertension, family history of hypertension and to educate women about the early warning signs of preeclampsia[16].

Majority of women undergo ultrasonography at around 18-20 weeks to rule out congenital anomalies. Placental localization in the same settings does

not add to the cost of screening procedure. Thus it is non-invasive, easy to perform, easily available, reliable and cheap.

# AIMS AND OBJECTIVES

To study the correlation of placental location on ultrasound and development of hypertensive diseases of pregnancy**.**

# REVIEW OF LITERATURE

Hypertensive disorders of pregnancy, including pre-eclampsia complicate up to 7-10% of all pregnancies[1], it constitutes one of the major causes of maternal and perinatal morbidity and mortality. Hypertensive disorders of pregnancy along with haemorrhage and infection constitute the deadly triad, which contribute greatly to maternal morbidity and mortality.

Hypertension is defined as either systolic BP of 140mmHg or greater, diastolic BP of 90 mmHg or greater or both. Hypertension is considered mild until diastolic and systolic levels are more than 110 mmHg and more than 160mmHg, respectively. It is recommended that a diagnosis of hypertension requires at least two BP readings which are at least 4 hours apart. Although all occasion especially when there is severe hypertension, the diagnosis can be confirmed within a shorter interval (even minutes) to facilitate timely antihypertensive therapy.

**Blood pressure Measurement**

Conventional mercury sphygmomanometer is the gold standard for blood pressure measurement.

The bell of stethoscope is used as it amplifies the Korotkoff sounds.

The optimal measurement of BP is made with the patient comfortably seated, legs crossed and the back and right arm supported so that the middle of the cuff on the upper arm is at the level of the right atrium (midpoint of the sternum).

The patient should be made to relax and not to talk during the measurement procedure; ideally 5 minutes should elapse before the first reading is taken.

Inflate the cuff above the systolic pressure and recognize the disappearance of the radial pulse.

The diastolic blood pressure is determined by Korotkoff (V) sound (disappearance of the sound).

If elevated on initial assessment, BP should be repeated after some time in attempt to eliminate spuriously elevated BP determinations.

The measurement of BP taken in the upper arm with the women in left lateral position will be lower BP recordings because the cuff will be above the heart.

Earlier known as pregnancy induced hypertension, HYPERTENSIVE DISORDER OF PREGNANCY covers a spectrum of conditions, of which preeclampsia poses the greatest potential risk and remains one of the most common causes of maternal death.

**CLASSIFICATION**:-

1. Gestational Hypertension:-

Sustained systolic blood pressure at or above 140 mmHg and a diastolic blood pressure of 90mmHg or greater after 20 weeks in the absence of accompanying proteinuria in a woman with previously normal blood pressure[17,18]. Increase in BP must be present on at least two separate occasions, 4 hours or more apart in pregnancy and hypertension resolves by 12 weeks postpartum.

2. Chronic Hypertension:-

Chronic hypertension is defined as a high blood pressure known to predate conception or detected before 20 weeks of gestation[17]. The blood pressure of more than systolic 140 and diastolic of 90 mmHg present before 20 weeks of gestation.

3. Preeclampsia Superimposed On Chronic Hypertension:-

Onset of features diagnostic of preeclampsia in women with chronic hypertension beyond 20 weeks of gestation. If new onset or worsening baseline hypertension is associated by new onset proteinuria, then the diagnosis of superimposed pre-eclampsia is made. It tends to be more severe and often accompanied by fetal growth retardation. If the only manifestation present is elevation in blood pressure to levels less than 160mm Hg systolic and 110 mm Hg diastolic and there is associated proteinuria, this is considered to be superimposed preeclampsia without severe features. The presence of organ dysfunction is the superimposed preeclampsia with severe features[17].

4. Preeclampsia and Eclampsia Syndrome:-

Preeclampsia is a syndrome which is found to include the development of new-onset hypertension in the second half of pregnancy. Although often accompanied by new onset proteinuria, preeclampsia can be associated with signs and symptoms, including visual disturbances, headaches, epigastric pain and rapid development of edema in severe form.

It is a pregnancy specific syndrome that is occurring due to the reduced organ perfusion which occurs secondary to vasospasm and endothelial activation.

**DIAGNOSTIC CRITERIA FOR PREECLAMPSIA[17]**

**Blood Pressure**

Systolic blood pressure of 140mmHg or more and the diastolic blood pressure of 90 mm Hg or more that is recorded on two occasions at least 4 hours apart after 20 weeks of gestation in women with previously normal blood pressure recordings[17].

Systolic blood pressure of 160 mm Hg or more and the diastolic blood pressure of 110 mm Hg or more. (Severe hypertension can be confirmed within short interval [minutes] to facilitate initiation of timely antihypertensive therapy)[17].

**Proteinuria**

- Dipstick reading of 2+(used only if other quantitative methods not available)
- Or in the absence of proteinuria , new-onset hypertension with the new onset of any of the following:
- 300 mg or more per 24 hour urine collection
- Protein/creatinine ratio of 0.3 mg/dl or more or
- Thrombocytopenia : Platelet count less than 1,00,000x10^9/L
- Renal insufficiency: Serum creatinine concentrations more than 1.1 mg/dl or serum creatinine concentration is doubled, which occurs in the absence of other renal disease.

- Impaired Liver function: Elevated blood concentrations of liver transaminases which occurs to twice the normal concentration.
- Pulmonary edema

  New – onset headache that is unresponsive to medication and not accounted for by alternative diagnoses or visual symptoms[17].

**Severe Features**[17]

- Systolic blood pressure of 160 mm Hg or more and the diastolic blood pressure of 110 mm Hg or more on two occasions at least 4 hours apart (unless antihypertensive therapy is initiated before this time)
- Thrombocytopenia (platelet count less than 100,000 X10^9/L)
- Impaired liver function that is indicated by abnormally elevated blood concentrations of liver enzymes (to twice the upper limit normal concentration), and severe persistent right upper quadrant or epigastric pain that does not respond to any medication and is not accounted for by any alternative diagnoses.
- Renal insufficiency (serum creatinine concentration that is more than 1.1 mg/dL or the serum creatinine concentration is doubled that occurs in the absence of other renal disease)
- Pulmonary edema
- New-onset headache that is unresponsive to medication and is not accounted for by any alternative diagnosis.
- Visual disturbances[17]

**ASSOCIATED FACTORS**

**Proteinuria:-**

It is the marker of glomerular damage. It reflects the glomerular damage which causes the leakage of proteins through the basement membrane.

Significant proteinuria is defined as proteinuria of 300mg/24 hours[17] or a protein to creatinine ratio of 0.30 or more[17,19] or persistent 30mg/dl (1+) in random clean catch sample on at least 2 occasions that is collected 6 hours

apart. If proteinuria is >5gm/24 hours or persistent 3+ dipstick or more the condition is called as severe preeclampsia[17].

**Theories Regarding The Causation Of Preeclampsia:-**

**Immunological**

The trophoblastic cells are found to express unique HLA-G enclosed class-1 MHC molecule which is allowing the trophoblast cells to invade deep into spiral arteries instead of usual class-I AND II MHC antigen. In the patients developing preeclampsia, there is occurrence of immune resistance to the invading trophoblast by the maternal immune system which is resulting in poor spiral artery remodeling that is a initially cited feature in preeclampsia. Preeclampsia is an immune system mediated condition, the microscopic changes that occur at the maternal placental interface are suggestive of acute graft rejection.

The risk of preeclampsia is increased when there is impairment of formation of blocking antibodies to the antigenic sites in the placenta or when there is increase in number of antigenic sites as in hyperplacentosis.

- T-lymphocyte secrete cytokines which lead to enhanced implantation and their dysfunction promote occurrence of preeclampsia. There is reduction of T helper cells in preeclampsia.
- Immunocytochemical analysis – VCAM (vascular cell adhesion molecule) is increased in preeclampsia.
- Antibodies to B2 glycoprotein I is more relevant immune complex and antiendothelial antibodies are the ones that might be involved in the pathogenesis.

One $CD4^+$ lymphocyte subset that might have been involved in the pathophysiology of preeclampsia is the $CD4^+CD25^+FoxP3^+$ (forkhead box P3) Treg (regulatory T cell). One of the Treg transcription factor is FoxP3[20]. It has been reported that there are reduced numbers of Tregs in preeclampsia compared with normal pregnancies. $CD4^+CD25^+FoxP3^+$ Treg function is reduced in preeclampsia, which may be related to the presence of inflammatory conditions. The Treg pool in preeclamptic women consists

mostly of $CD4^+CD25^+FoxP3^+HLA\text{-}DR^+CD45RA^-$ cells. Although these cells express HLA-DR, which is responsible for suppression activity, they exhibit reduced regulatory capacity. Thus, Tregs and Treg subsets seem to play a role in the pathophysiology of preeclampsia[20].

In addition to Tregs, $CD4^+$ IL-17-producing T cells (Th17) also participate in pathology of preeclampsia. Preeclamptic patients have been found to have a lower ratio of Tregs: Th17 cells. Decreased mRNA levels of FoxP3 and increased levels of the Th17 transcription factor RORc and the Th1 transcription factor T-bet are found in preeclamptic women compared with healthy pregnant women. The predominance of Th17 cells in preeclampsia, accompanied by decreased Treg function and an altered balance in the Th17:Treg ratio, might be a result of altered levels of cytokines, including IL-6 and IL-1, which promote differentiation of these cells from progenitor cells. However, $CD8^+$ lymphocytes and NK cells also secrete IL-17 which might also contribute to inflammation in this syndrome[20].

T lymphocytes are also possessing functional RAS (renin angiotensin system) elements, able to produce AT2 (angiotensin-2) at inflammatory sites. As AT2 (angiotensin-2) can promote chemotaxis of NK cells and T cells by binding to AT1-R(angiotensin 1 -receptor), a RAS-mediated inflammatory pathway may also be involved in preeclampsia[20].

**Genetic Susceptibility**

Preeclampsia has about 31% estimated genetic inheritance.

Pregnancy induced hypertension is inherited both by single gene and by the multifactorial inheritance.

Modes:-

- Simple recessive model with the genes in mother(chromosome 1,3,9)
- Dominant model that is with incomplete penetration.
- HLA DR4- The transmission from mother to daughter is through the HLA DR4.
- Angiotensinogen gene variant T235- There is high incidence of pregnancy induced hypertension.

- Factor V laden mutation polymorphism.
- The Epigenetic Hyperhomocystinaemia which is found to be associated with methyltetrahydrofolate homozygozity.
- Imprinting that is involved in the pathogenesis of preeclampsia susceptibility locus on chromosome 10q 22-1.
- The eNOS gene susceptibility which occurs in the region of chromosome 7q36.
- Occurrence of Polymorphism of the TNFα and Gene-mediating endothelial dysfunction, lymphotoxins, ILi.

**Placenta and Preeclampsia**

Placenta is the vital link between the mother and the fetus for metabolic exchange, endocrine and other body functions.

Hypoperfusion of the placenta produces various factors which can injure the endothelial cells. Circulating factors which interfere with the action of vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) that are responsible for playing a major role in the maternal manifestation of this disorder.

The shallow placentation that occur in preeclampsia is mainly due to the inability of trophoblasts to invade the decidual vessels. A subset of cytotrophoblasts is found to migrate through the placental implantation site normally and invades the decidua's tunica media of maternal spiral arterioles and replaces its endothelium that is psuedo vascularization. Due to these changes, there is transformation of small muscular arterioles to large capacitance, low resistance vessels. This facilitates the blood flow to the maternal fetal interface. Remodeling of these arterioles is found to begin in the first trimester and ends at around 18-20 weeks of gestation.

In preeclampsia, the invasion of the decidual arterioles is not completely occurring. The tunica media is not invaded by the cytotrophoblasts that results in mostly intact arterioles which are capable of vasoconstriction. Histological evaluation of the placental bed have been found to demonstrate very few cytotrophoblasts which go beyond the decidual layer. The

trophoblastic differentiation that occurs along the invasive pathway is involving alteration in the expression of a number of different classes of molecules including cytokines, adhesion molecules, extracellular matrix, metalloproteinases and the class Ib major histocompatibility complex molecules, HLA-G Angiogenesis is critical for successful placentation.

The development of preeclampsia may involve several factors including persistent hypoxia at the placental level and the release of high amounts of syncytiotrophoblast microvesicles (STBM's). These activate monocytes, DC's, NK cells and neutrophils which promote persistent inflammatory conditions in this syndrome[21].

Angiogenesis is facilitated by interaction of the VEGF receptor family with both vascular endothelial growth factor (VEGF) and placental growth factor (PlGF). In pregnancy, the serum level of PlGF is raised significantly. There is reduction in serum level of both VEGF and PlGF in women who develop preeclampsia. The angiogenic factors for example, the placental growth factor, soluble fms-like tyrosine kinase 1 and soluble endoglin, all originate somehow from the syncytiotrophoblast, are biomarkers that have predictive potential for preeclampsia and it's related adverse outcomes[22].

Poor extravillous trophoblastic invasion and arterial remodeling is the underlying pathology in preeclampsia[23]. The disease is typically consists of 2 phases. First, the placental phase characterized by compromised trophoblast invasion and uterine arterial remodeling .Second, the maternal phase characterized by wide-spread maternal endothelial damage and dysfunction, which results from a variety of toxic products that are released from a hypo-perfused, hypoxic placenta. These products might include lipid peroxidases, syncytiotrophoblast microvillous fragments, and inflammatory chemokines[23].

Uniform distribution of the placental blood supply is not present. The site of implantation of placenta and resultant location of placenta within the uterus are likely important predictors of placental blood flow and therefore the pregnancy outcome. The two uterine arteries have a significant number of

branches and each branch supplies the corresponding side of the uterus. In the patients with centrally located placenta both the uterine arteries demonstrate similar resistance and the uteroplacental blood flow are met equally by both of the uterine arteries.

When the placenta is located laterally, the uteroplacental blood flow is met with one of the uterine artery along with some contribution from the other uterine artery through the collateral circulation.[1] The degree of collateral circulation may not be the same in all subjects and deficient contribution may facilitate the development of preeclampsia[1].

The significance of normal placentation for this cytotrophoblast fail to adapt a vascular adhesions phenotype in preeclampsia.

**Endothelial Dysfunction**

Endothelial dysfunction resulted from the generalized perturbation of normal maternal intravascular inflammatory adaptation to pregnancy.

There is immunologically mediated deficiency in the trophoblastic invasion of the spiral artery that causes the release of a number of factors into maternal circulation. This causes the activation of vascular endothelium. This damaged endothelium activated endothelial cells are responsible for promoting coagulation and increased sensitivity to vasopressor agents.

Endothelial dysfunction is increased in preeclampsia due to the associated reduction of unbound serum level of angiogenic factors such as VEGF and PlGF that occurs due to the upregulation of the soluble fms like tyrosine kinase receptor and serum level of sFltl .

**Prostaglandins**

In preeclampsia, there is imbalance between PGI2 (Prostacycline) and TxA2 (Thromboxane A2). TxA2 is increased. The trophoblastic hypo-invasiveness and increased peripheral vascular resistance in the pregnant mother have been attributed to the decreased levels or responses to PGE2[23].

**Endothelins**

Higher levels of Endothelin 1 is found in preeclampsia. These are polypeptide with significant vasoconstricting property.

ET1 (endothelin-1)is being produced by vascular endothelial cells within the placenta and the decidua. ET1 action on EVT (extravillous trophoblast) cells has shown to follow the PLC-dependent elevation of [Ca2+] and activation of MAPK. PGE2, a major decidual product, has been shown to stimulate EVT cell migration by its binding to PGE receptors, in particular EP1 and EP4. EP1-mediated migratory responses resulted from an increase in [Ca2+] because of release from intracellular storage sites, and an activation of the calpain family of [Ca2+] dependent proteases, required for cell detachment from the substratum. In preeclampsia, trophoblast's hypo-invasiveness and increased peripheral vascular resistance in the pregnant mother has been ascribed to decreased levels or responses to PGE2[23].

**Nitric Oxide**

There is decreased endothelial nitric oxide synthase production. There may be increased production of nitric oxide as a compensatory mechanism. It is a consequence of hypertension and not the cause of hypertension.

Ionic calcium is crucial for the synthesis of vasosubstances in the endothelium like PGI2 and nitric oxide. An alteration in the action of nitric oxide may be related to inactivation by the free radical superoxide (secondary to inflammatory response).

**Oxidants and Antioxidants**

Increased free radical in preeclampsia, free radical causes endothelial damage. Oxidants, that are generated by the activated neutrophil NADPH oxidase react with different targets to form toxic metabolites which are the product of lipid peroxidation such as 4-hydroxynoneal that contribute the microbial death and damage induced by the neutrophils. Lipid peroxidation is elevated in preeclampsia suggesting that these women are under persistent oxidative stress that contributes to the inflammatory response[20].

**Placental Proteins**

Corticotrophin releasing factor, HCG, Activin A and inhibin A are said to play role in pathogenesis of Pregnancy induced hypertension.

**Vasospasm**

Vasospasm results in the endothelial damage and inter endothelial cell leakage which resulted in subendothelial deposition of fibrin and platelets and hence uteroplacental insufficiency.

**PATHOPHYSIOLOGY**

Several mechanisms of disease have been proposed in preeclampsia including the following:

- Chronic uteroplacental ischemia
- Increased trophoblast apoptosis or necrosis
- Immune maladaptation
- Genetic imprinting
- Very low-density lipoprotein toxicity
- An exaggerated maternal inflammatory response to deported trophoblasts[20,24].

More recent observations suggest a possible role for imbalances of angiogenic factors in the pathogenesis of pre-eclampsia. It is possible that a combination of some of these purposed mechanisms may be responsible for triggering the clinical spectrum of preeclampsia. Clinical and experimental evidence suggest that uteroplacental ischemia leads to increased circulating concentrations of antiangiogenic factors and angiogenic imbalances.

**Vascular Changes**

Interaction of various vasoactive agents, such as prostacyclin (vasodi lator), thromboxane A2 (potent vasoconstrictor), nitric oxide (potent vasodilator), and endothelins (potent vasoconstrictors) results in another significant change described in preeclampsia: intense vasospasm. In a study that was using invasive hemodynamic monitoring in women with preeclampsia, it was found that before intravenous fluid therapy, women with preeclampsia

had hyperdynamic ventricular function with low pulmonary capillary wedge pressure[25], but after aggressive fluid therapy, the pulmonary capillary wedge pressure increased significantly above normal levels[25] with increasing risk of pulmonary edema.

**Hematological Changes**

- Thrombocytopenia results from increased platelet activation, aggregation, and consumption[26] and is a marker of disease severity.
- A platelet count less than 150,000 x 109/L is found in approximately 20% of patients with preeclampsia, varying from 7% in cases without severe manifestations to 50% in cases with severe manifestations[27].
- While Interpretating hematocrit levels in preeclampsia, it should be considered that hemolysis and hemoconcentration may occur[28].
- Erythrocytes contain high concentration of Lactate dehydrogenase. High serum concentrations of LDH (more than 600 IU/L) may indicate hemolysis[29,30].

**Hepatic Changes**

- Alanine aminotransferase and AST may be elevated.
- Aspartate aminotransferase is the dominant transaminase released into the peripheral circulation in liver dysfunction due to preeclampsia and is related to periportal necrosis. AST is elevated to a greater extent than ALT, at least initially, might help in distinguishing preeclampsia from other potential causes of parenchymal liver disease that have ALT usually higher than AST.
- The elevated serum levels of LDH in preeclampsia are due to hepatic dysfunction (LDH derived from ischemic, or necrotic tissues, or both) and hemolysis (LDH from red blood cell destruction).
- Alterated hepatic synthetic function, is usually indicated by abnormalities of prothrombin time, partial prothrombin time, and fibrinogen, usually develop in advanced preeclampsia.

- Evaluation of these coagulation parameters is only beneficial when the platelet count is below 150,000 x 109/L, there is significant liver dysfunction, or there is suspected placental abruption[31].

**Renal Changes**

- The histopathologic renal changes that are particularly described in preeclampsia as glomerular endotheliosis which consists of swollen, vacuolated endothelial cells with fibrils, swollen mesangial cells, subendothelial deposits of protein reabsorbed from the glomerular filtrate, and tubular casts[32,33].
- Proteinuria in preeclampsia is nonselective, as it occurs due to the increased tubular permeability to most large molecular-weight proteins (albumin, globulin, transferrin, and hemoglobin).
- Urinary calcium decreases because of an increased tubular reabsorption of calcium.
- In preeclampsia, there occurs contraction of the intravascular space secondary to vasospasm causing worsening renal sodium and water retention[34].
- Preeclampsia with severe features might involve the acute renal deterioration as part of the clinical spectrum. Oliguria in severe preeclampsia is due to intrarenal vasospasm with about 25% reduction in glomerular filtration rate.
- In these patients, transient oliguria (less than 100 mL over 4 hours) is a common observation in labor or the first 24 hours of the postpartum period.
- Plasma concentrations of uric acid concentration increases to a greater extent in preeclampsia[35]. The most common accepted explanation for hyperuricemia in preeclampsia is that besides increased production, there is increased reabsorption and decreased excretion of uric acid in the proximal renal tubules.

**Fetal Consequences**

Due to the impaired uteroplacental blood flow that occur secondary to failure of physiologic transformation of the spiral arteries or placental vascular insults, or both, manifestations of preeclampsia also may be seen in the fetal–placental unit[36].

There occurs abnormalities in the placental bed and subsequent failure of physiologic transformation of the spiral arteries in the first or early second trimester limiting[37,38] the blood flow to the uteroplacental unit.

Additional mechanisms for chronic uteroplacental ischemia include placental vascular insults[39,40]. Among women with preeclampsia, clinical manifestations that follow from this uteroplacental ischemia include fetal growth restriction, non-reassuring fetal status, oligohydramnios, placental abruption, and demonstrated on antepartum surveillance. Consequently, fetuses of women with preeclampsia are at increased risk of spontaneous or indicated preterm delivery.

In a study conducted by Vidhu V. Nair et al., the frequency of the central placenta was 377 (83.8%) and frequency of lateral placenta was 73 (16.2%). Central placenta group had an abnormal outcome in 182 (48.3%) and the lateral placenta group with abnormal outcome were 44 (60.3%). The abnormal maternal outcomes like hypertensive disorders (33.3%), intrauterine growth restriction (10.2%), antepartum hemorrhage (25%), preterm birth (16.3%) were found to be more in group with lateral placenta. The number of neonates of women in central placenta group having NICU admissions were 62 (16.4%) and neonates who had NICU admissions in the women with lateral placenta group were 19 (26%). Thus it was concluded from this study that there was significant association between lateral placentation and abnormal pregnancy and neonatal outcomes. Second trimester ultrasound can be used as non-invasive predictor of adverse pregnancy outcome and neonatal outcomes[41].

Similar study was conducted by Shailesh Janardhan Kore, to find out whether placental laterality as determined by ultrasound done between 20-24 weeks could be used as a predictor for development of preeclampsia. It

was found that out of 200 women, 161 women had central placenta (Group -A) and 39 had unilateral placenta (Group -B). Total of 32 women developed preeclampsia out of which 19(59.38%) had unilaterally located placenta at around 20-24 weeks. This relationship was statistically highly significant ($p<0.0001$). Using placental laterality as a screening test it was found that the sensitivity, specificity, positive predictive value and negative predictive value were 59.38%, 88.10%, 48.72 % and 91.93% respectively. It was concluded that placental laterality as determined by USG at 20-24 weeks was a simple yet reliable and cost effective predictive screening test for the development of preeclampsia[42].

In a similar study conducted by Ramya Kaku et al., to find out whether lateral location of placenta as seen on ultrasound at II-III trimester of gestation could be used to predict development of preeclampsia. It was found that 66% patients with lateral placenta had preeclampsia. In the central placenta group 36% developed preeclampsia. In this study, it was found that the association of lateral placenta as a predictor of preeclampsia had a P-value of <0.001 which is statistically significant. Primigravidas had higher incidence of preeclampsia as compared to multigravidas. Most of the preeclamptics had their onset at 29-32 weeks of gestation. It was concluded that placental position determined by ultrasound in II-III trimester of gestation was an excellent screening tool for the prediction of preeclampsia. This test was simple, non-invasive, cost -effective and convenient to the women[1].

In a study conducted by Ambika Rai et al from November 2013 to October 2015 which was done to assess the relationship between second trimester placental position by ultrasound and the subsequent development of preeclampsia. Out of the 106 patients studied, 69 had central placenta and 37 had lateral placenta. A total of 17 patients developed preeclampsia of which 12 had lateral placenta. This result was statistically significant with a p value of 0.001, it was concluded that increased association of development of preeclampsia in patients with laterally located placenta

which aided to the preventive approach and optimum management of preeclampsia[43].

In a study conducted by Anjali Gupta et al to evaluate the association of laterally located placenta on ultrasound with development of preeclampsia. It was found that out of 200 antenatal women, 84 had lateral location of placenta and 116 had central location of placenta. Amongst, 84 women with lateral placenta, 55 women (65.5%) developed preeclampsia and amongst 116 (58%) women with central placenta, 28 women (24.1%) developed preeclampsia. It was concluded that women with the laterally located placenta by ultrasound at 18-24 weeks of gestation have higher risk of developing preeclampsia[44].

In a study conducted by Alexander D. Kofinas et al on 153 pregnant women with normal pregnancies and 147 women with complicated pregnancies to evaluate the association of placental location and development of preeclampsia, intrauterine growth retardation and uterine artery resistance. The uterine artery resistance was determined by continuous wave Doppler flow velocity analysis and placental location determined by real time ultrasonography. In the presence of preeclampsia or intrauterine growth retardation up-to 75% of patients had unilaterally located placenta and 25% central placenta whereas it was found that if there was absence of preeclampsia and intrauterine growth retardation ,only 51% of patients had unilateral and 49% central placenta ($p<0.02$). In the patients with unilateral placenta, incidence of preeclampsia and intrauterine growth retardation was 2.8 fold and 2.7 fold higher than in patients with central placenta ($p<0.03$ and $p<0.01$) respectively. It was found that the unilateral placental location was more commonly associated with abnormal uterine artery flow velocity waveforms than central placental location ($p<0.001$). It was found that unilateral placental location may predispose to the development of preeclampsia and intrauterine growth retardation by its effect on uterine artery resistance[45].

In a study conducted by Sandhya K et al to study whether ultrasonically identified, placental laterality could be used as a predictor for development of preeclampsia. It was found that out of the total 300 women, 168(56%) had laterally located placenta and out of these, 112 (66.6%) developed preeclampsia, out of the remaining 112 (44%) had centrally located placenta and of them, 48 (36.3%) developed preeclampsia. The overall risk of developing preeclampsia with laterally located placenta was found to be 5.09 (odds ratio) and 95% confidence interval (2.40 to 10.88). The difference was found out to be statistically significant, p value (0.00002) by chi-square test. It was concluded that females with laterally located placenta determined by USG at 18 to 24 weeks of gestation had five times greater risk of developing hypertension. By the identification of such patients appropriate treatment could be initiated and the patients are regularly followed up[46].

In a study done by Alpana Singh et al to find out whether the placental laterality as a predictor of preeclampsia and small for gestational age baby, it was found that out of 347, a total of 270 patients were analyzed, 39 (14.4%) had lateral placenta and out of them 17 (43.5%) developed preeclampsia and 24 (61.5%) women had small for gestational age baby ($p<0.001$). In this study, 231 (85.5%) had central placenta and among them 49 (21.2%) developed preeclampsia and 63 (27.2%) had small for gestational age baby ($p<0.001$). It was concluded that laterally located placenta had significant association with preeclampsia and small for gestational age babies[47].

In a study conducted by Avik Ghosh et al to assess the relationship between the laterality of placenta by mid-trimester ultrasonography and perinatal outcome. It was found that out of total 300 women, 195/300 (65%) had central placenta and 105/300 (35%) had lateral placenta There were 4 IUFDs (1.33%); 2(1.03) had central placenta and 2 (1.9) had lateral placenta. Among 31 (10.33%) IUGR babies 13 (6.67%) were associated with central placenta and 18 (17.14%) with lateral placenta. Among 27 LBW

babies 16 (8.21%) were related to central placenta and 11 (10.48%) related to lateral placenta. It was found that laterally located placenta was associated with relatively poor perinatal outcome compared to central placenta with increased rates of IUGR, IUFD and LBW babies[48].

In a study conducted by Parul S. Jani et al to study, ultrasonically identified; placental laterality and uterine artery resistance could be used as a predictor for development of preeclampsia. Amongst, 400 cases, 80 (20%) cases had lateral placenta on ultrasound examination done at 18-24 weeks of gestation. Among, 80 women with laterally located placenta, 28 (35%) developed preeclampsia. This relationship is found to be statistically significant. Out of all 80 patients with lateral placenta, 26 had raised uterine artery resistance and out of those 26 patients, 22 developed preeclampsia and 54 women had no change in uterine artery resistance. Out of, 54 only 6 developed preeclampsia, that was statistically significant ($p<0.001$). It was found that the sensitivity of determining uterine artery resistance as a predictive test was found to be 84.6%, the specificity was 88.8%, positive predictive value was 78.5% and negative predictive value was 92.3%. It was concluded that preeclampsia is more in patients with lateral placenta but its sensitivity and specificity increases significantly when it is combined with uterine artery velocimetric waveform study and preeclampsia can be predicted in patients who have lateral placenta and raised uterine artery resistance[49].

In a similar study by Vandana Ambastha et al which was conducted to assess the incidence of preeclampsia and other feto-maternal outcome with centrally located placenta and those with laterally located placenta. It was found that out of total 250 women, 148 had central placenta in which 8(5.41%) developed preeclampsia, however 102 had lateral placenta in which 40(39.22%) developed preeclampsia. Thus in total 48 women developing preeclampsia, 40 (83.33%) women had lateral placenta whereas 8(16.67%) women had central placenta. The difference was found out to be statistically significant as p value is <0.001. It was concluded that

women with laterally located placenta determined by USG have 5 times greater risk of developing preeclampsia[50].

In a similar study conducted by Bhattacharjee Ak et al on 100 pregnant women with lateral lying placenta and 100 pregnant women with centrally located placenta to assess the correlation of placental location and development of preeclampsia. It was found that out of the 100 women with laterally located placenta, 66(66%) developed preeclampsia, while 36 women (36%) out of the remaining 100 women with centrally located placenta developed preeclampsia. It was found that preeclampsia was five times greater in females with laterally located placenta as compared to those with centrally located placenta (p 0.01). It was concluded that presence of lateral placenta predisposed women to develop preeclampsia more frequently and location of placenta could be used as an indicator for development of preeclampsia[51].

In a study conducted by Madhu Patni et al, to study whether location of placenta sonographically can be used as a screening tool for preeclampsia and IUGR. Out of the 80 total cases, it was found that the maximum cases were found in age group 21-25 years in both the groups. Out of the 40 cases only one case was found to develop eclampsia in group A whereas no such case was found in group B. It was concluded that detection of placental site during routine ultrasonography is very simple, doesn't need an expert radiologist and can be done even by obstetrician[52].

In a study conducted by Ananya Priyadarshini et al which was conducted to determine the incidence of lateral location of placenta and to study the relationship between placental location and development of preeclampsia. Out of the total 100 cases, there were 68 cases of lateral placenta and 32 cases of central placenta. Most of the cases belonged to middle class living in the urban area. It was concluded that 48.5% patients with lateral placenta and 46.9% with central placenta developed preeclampsia. There was significant association was found between laterality of placenta and

development of PIH. The laterally located placenta is associated with increased risk of developing preeclamsia[53].

In a study conducted by Elham Keshavarz to find the blood supply distribution within the uterus which is not similar in central compared to the lateral side, thus site of implantation and resulting location of placenta are likely to have a profound effect on the pregnancy outcome. Out of the total 379women, 121 females with third degree of preeclampsia were considered as cases and 258 females with normal pregnancy were the controls. It was found that pregnancies complicated by the preeclampsia were more commonly associated with lateral placentation in the second trimester when compared with the non-preclamptic pregnancies. It was concluded that the placental location that could be easily assesses at the middle trimester of pregnancy by routine screening ultrasonography, is an ideal predictive test for evaluating risk of development of hypertension[54].

# MATERIAL AND METHODS

The study was performed in the Department of Obstetrics and Gynecology, Dayanand Medical College and Hospital, Ludhiana which is a tertiary care hospital.

This was a prospective study over a period of 1 year from January 2019 to December 2019 where pregnant women at 18-24 weeks are followed for the development of hypertension and the association of hypertension with lateral placenta is studied.

METHODS OF COLLECTION OF DATA

Study population included indoor as well as outdoor patients. A structured predesigned proforma was used. Detailed history including basic demographics such as age, weight, height, socioeconomic status B.P. were taken. Relevant antenatal history and routine investigations were recorded. Relevant past history and family history were taken. The results obtained were followed up and evaluated.

INCLUSION CRITERIA

All pregnant patients between 18-24 weeks of gestation.

Patients consenting to participate in the study.

EXCLUSION CRITERIA

Subjects with concurrent diseases like:-

Autoimmune diseases.

Lupus anticoagulant

Anticardiolipin antibody

Renal diseases

Connective tissue diseases

Thyrotoxicosis

Uterine anomalies

Substance abuse

- Alcohol
- Drug addiction

OUTCOME:-

Correlation of placental location and development of hypertensive disease of pregnancy and it's evaluation in terms of maternal outcome at the end of pregnancy.

STATISTICAL ANALYSIS

Data was described in terms of range, mean +/- standard deviation (SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. For comparing categorical data, Chi square (X*2) test was performed and exact test is used when the expected frequency is less than 5. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations are done using SPSS (Statistical Package for the Social Science) SPSS21 version statistical program for Microsoft Windows.

THE STUDY DID NOT REQUIRE ANY INVESTIGATION OR INTERVENTION TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMAL.

ETHICAL CLEARANCE HAS BEEN OBTAINED FROM THE INSTITUTION

## OBSERVATION AND RESULT

In our study total of 100 cases were taken.

Group A- women with lateral placenta.

Group B- women with central placenta.

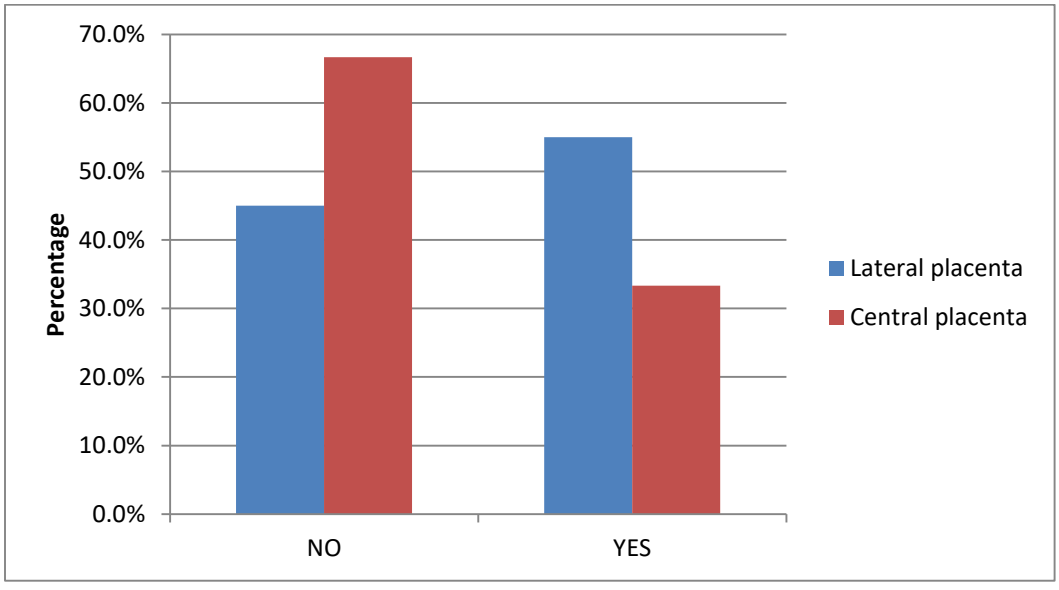
Group C- women with lateral placenta who developed hypertension.

Group D- women with central placenta who developed hypertension.

Table 1. The distribution of women according to the location of placenta and development of hypertension.

| | | Group A | | Group B | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| HTN | NO | 18 | 45.0% | 40 | 66.7% | 58 | 4.625 | 0.032 |
| | YES | 22 | 55.0% | 20 | 33.3% | 42 | | |

Figure 1. The distribution of women according to the location of placenta and development of hypertension.

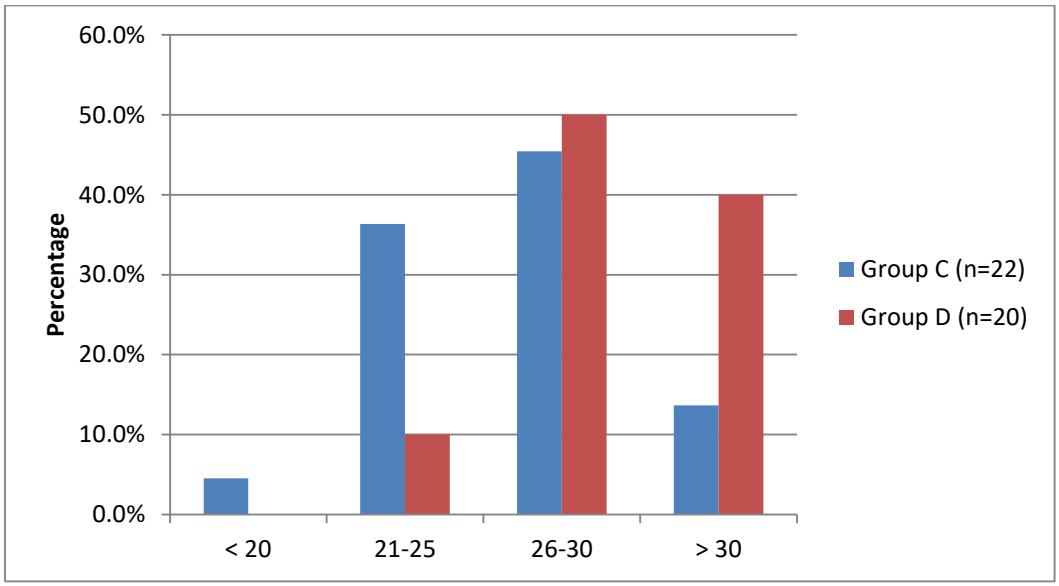



In our study in group A with lateral location of placenta, 55% (n=22) developed hypertension and in group B with central location of placenta 33.3% (n=20) developed hypertension.

Table 2. The distribution of women under study who developed hypertension according to age.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| Age group | < 20 | 1 | 4.5% | 0 | 0.0% | 1 | 0.931 | 0.335 |
| | 21-25 | 8 | 36.4% | 2 | 10.0% | 10 | 4.014 | 0.045 |
| | 26-30 | 10 | 45.5% | 10 | 50.0% | 20 | 0.086 | 0.768 |
| | > 30 | 3 | 13.6% | 8 | 40.0% | 11 | 3.787 | 0.052 |

Figure 2. The distribution of women under study who developed hypertension according to age.




In our study in group C, women with age <20 years-4.5% (n=1) developed hypertension, with age 21-25 years -36.4% (n=8) developed hypertension, with age 26-30 years-45.5% (n=10) developed hypertension and age >30 years 13.6% (n=3) developed hypertension.

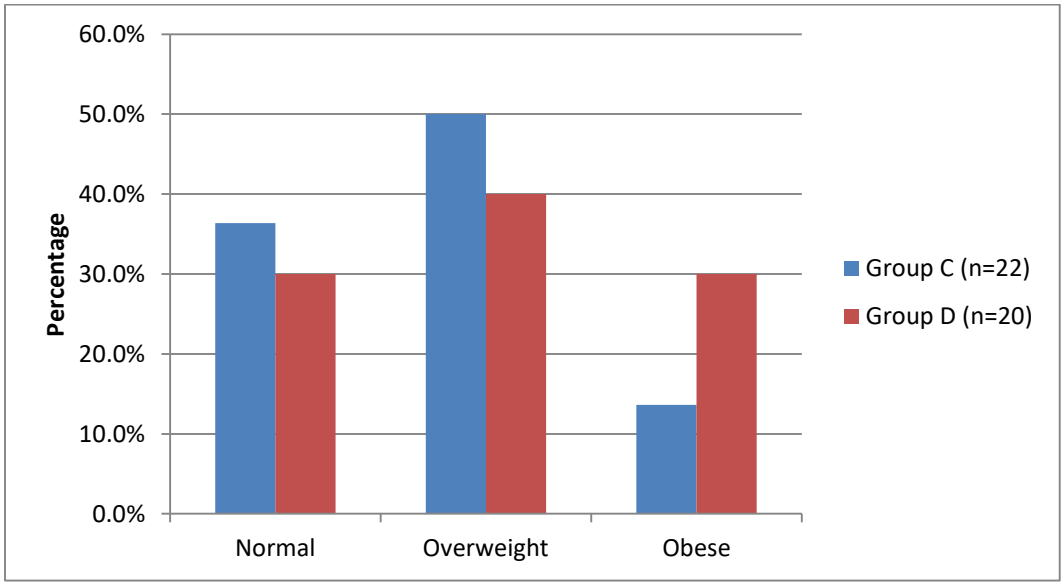
In group D, women with age <20 years, no patient developed hypertension, with age 21-25 years -10% (n=2) developed hypertension, with age 26-30 years 50% (n=10) developed hypertension and age >30 years 40% (n=8) developed hypertension.

In our study the majority of patients who developed hypertension in the group C and D belonged to age group of 26-30 years.

Table 3. The distribution of women who developed hypertension according to BMI

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| BMI group | Normal | 8 | 36.4% | 6 | 30.0% | 14 | 0.190 | 0.662 |
| | Overweight | 11 | 50.0% | 8 | 40.0% | 19 | 0.423 | 0.516 |
| | Obese | 3 | 13.6% | 6 | 30.0% | 9 | 0.166 | 0.197 |

Figure 3. The distribution of women who developed hypertension according to BMI




In our study the group C, 36.4% (n=8) women with normal weight, 50% (n=11) overweight women and 13.6% (n=3) obese women developed hypertension.

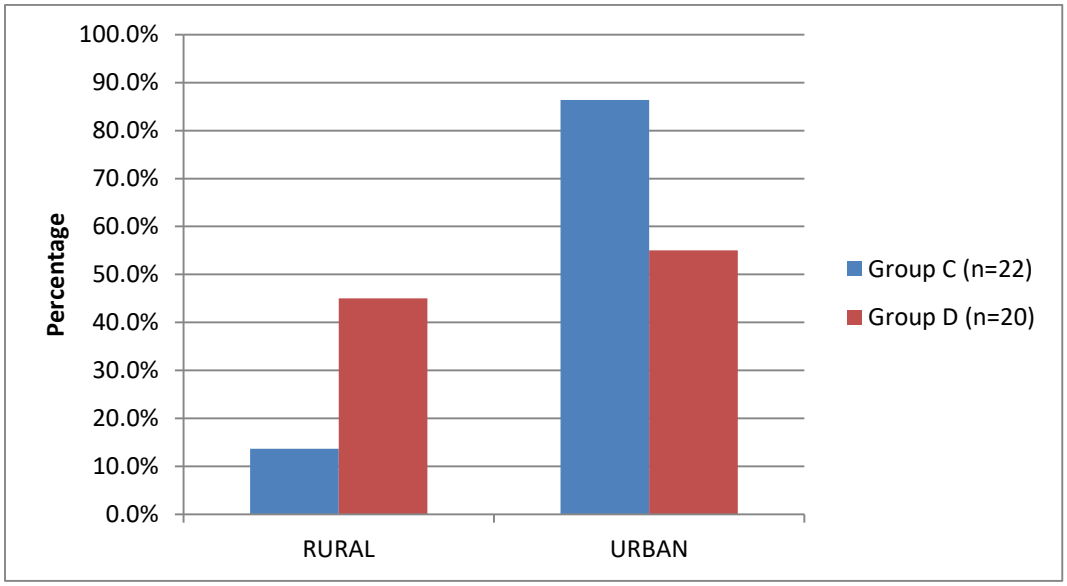
In the group D, 30% (n=6) normal weight women, 40% (n=8) overweight women and 30% (n=6) obese women developed hypertension.

In our study the women in group C and group D who developed hypertension were overweight.

Table 4. The distribution of women who developed hypertension according to residential area.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-Square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Area | Rural | 3 | 13.6% | 9 | 45.0% | 12 | 5.049 | 0.024 |
| | Urban | 19 | 86.4% | 11 | 55.0% | 30 | | |

Figure 4. The distribution of women who developed hypertension according to residential area.




In our study in group C, 13.6% (n=3) in rural area and 86.4% (n=19) in urban area developed hypertension.

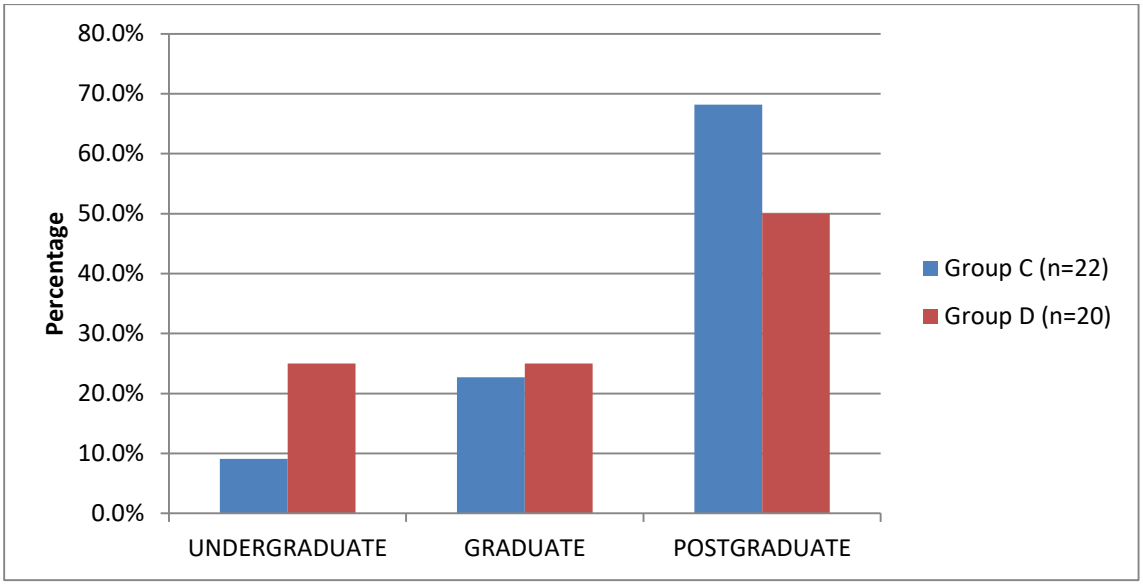
In group D, 45% (n=9) in rural area and 55% (n=11) in urban area developed hypertension.

In our study majority of women in both the groups who developed hypertension belonged to urban area.

Table 5. The distribution of women who developed hypertension according to education

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Education | Undergraduate | 2 | 9.1% | 5 | 25.0% | 7 | 1.909 | 0.167 |
| | Graduate | 5 | 22.7% | 5 | 25.0% | 10 | 1.616 | 0.204 |
| | Postgraduate | 15 | 68.2% | 10 | 50.0% | 25 | 1.437 | 0.231 |

Figure 5. The distribution of women who developed hypertension according to education




In our study in group C, 9.1% (n=2) undergraduate women, 22.7% (n=5) postgraduate and 68.2% (n=15) postgraduate developed hypertension.

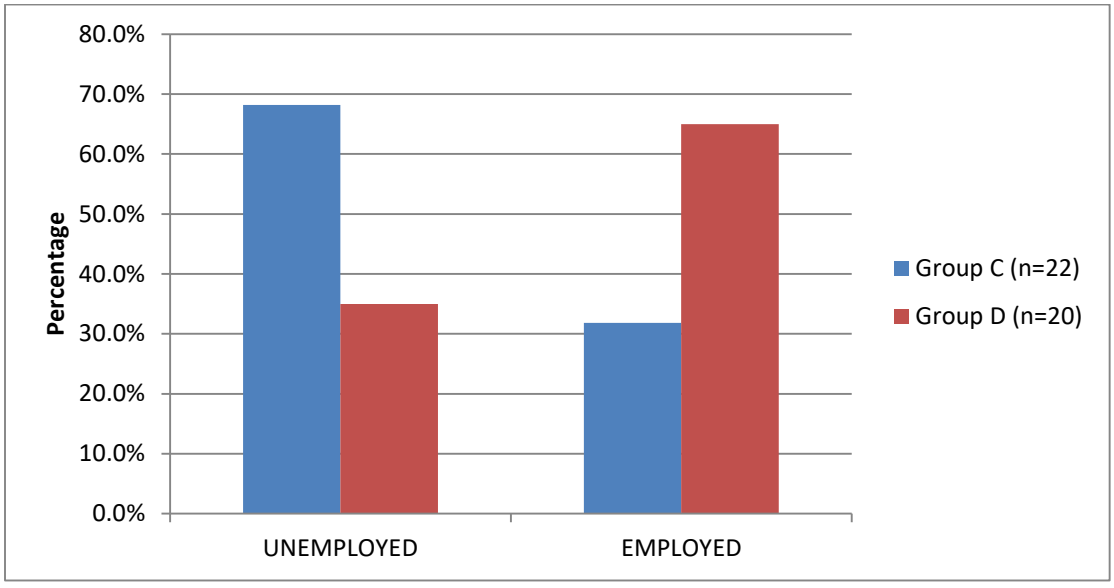
In the group D, 25% (n=5) undergraduate women, 25% (n=5) graduate and 50% (n=10) postgraduate developed hypertension.

In our study, women in group C and group D who developed hypertension belonged to postgraduate.

Table 6. The distribution of women who developed hypertension according to occupation.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Occupation | Homemaker | 15 | 68.2% | 7 | 35.0% | 22 | 4.624 | 0.031 |
| | Employed | 7 | 31.8% | 13 | 65.0% | 20 | | |

Figure 6. The distribution of women who developed hypertension according to occupation.

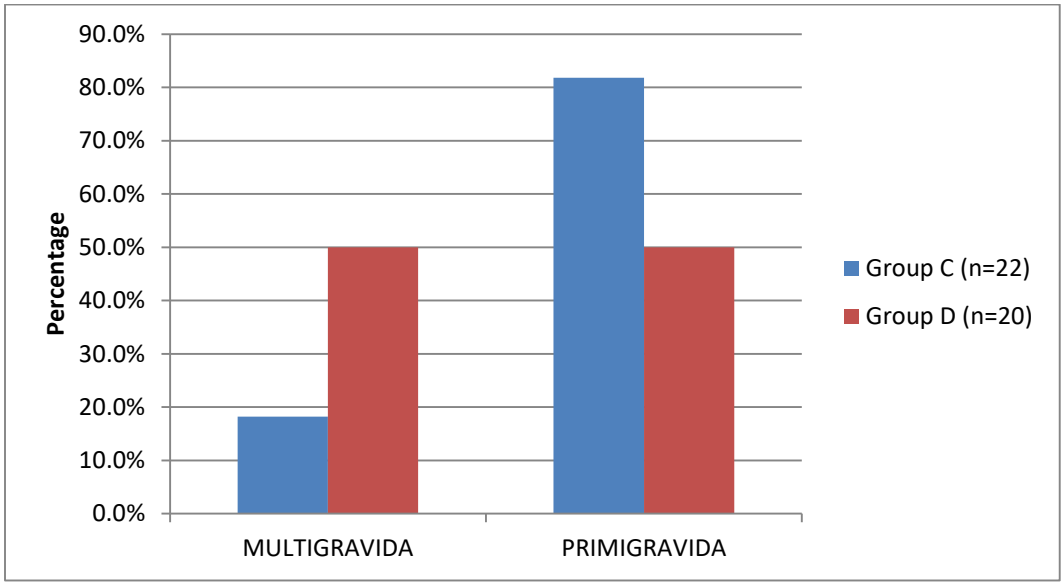



In our study in group C, 68.2% (n=15) were homemaker/housewife and 31.8% (n=7) were employed whereas in group D, 35%(n=7) were housewife/homemaker and 65%(n=13) were employed.

Table 7. The distribution of women who developed hypertension according to parity.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-Square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Parity | Multigravida | 4 | 18.2% | 10 | 50.0% | 14 | 4.773 | 0.029 |
| | Primigravida | 18 | 81.8% | 10 | 50.0% | 28 | | |

Figure 7. The distribution of women who developed hypertension according to parity.




In our study in group C, 18.2% (n=4) multigravida and primigravida 81.8% (n=18) women developed hypertension.

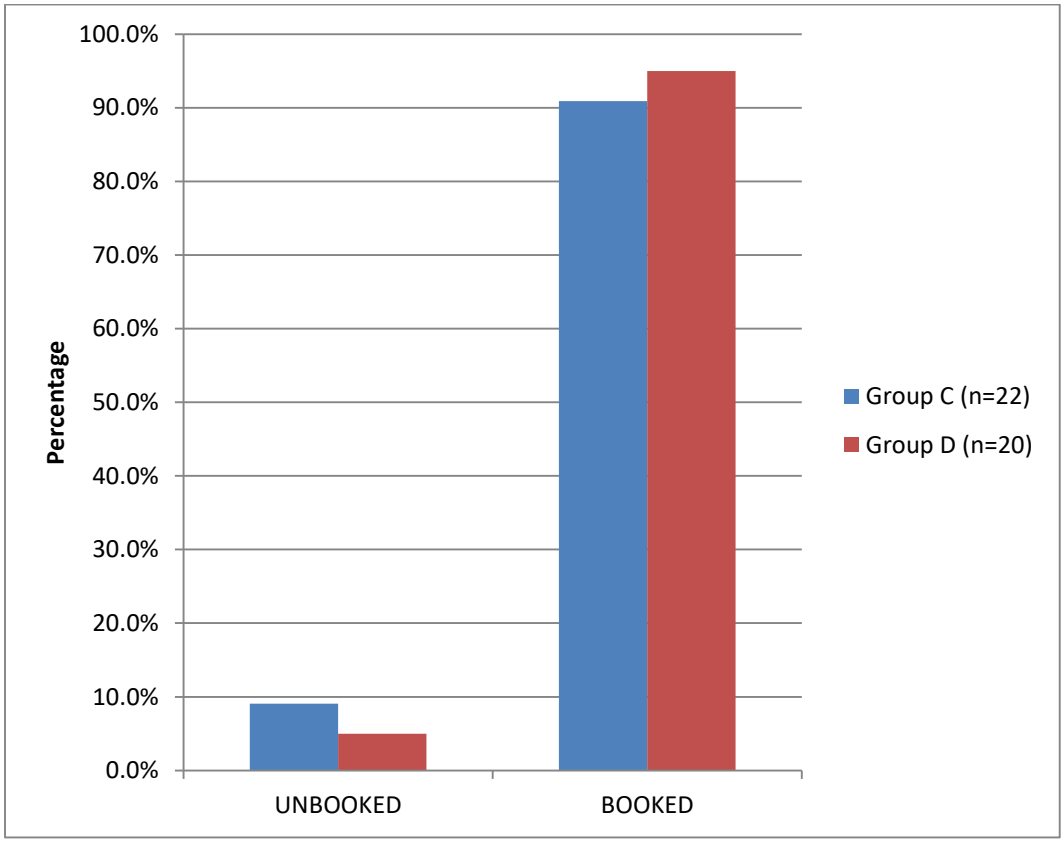
In group D, 50% (n=10) multigravida and 50% (n=10) primigravida women developed hypertension.

In our study the percentage of women in group C who developed hypertension was more among primigravida and group D who developed hypertension was equal among multigravida and primigravida.

Table 8. The distribution of women who developed hypertension according to antenatal care.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-Square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Booked/ unbooked | Unbooked | 2 | 9.1% | 1 | 5.0% | 3 | 0.264 | 0.607 |
| | Booked | 20 | 90.9% | 19 | 95.0% | 39 | | |

Figure 8. The distribution of women who developed hypertension according to antenatal care.

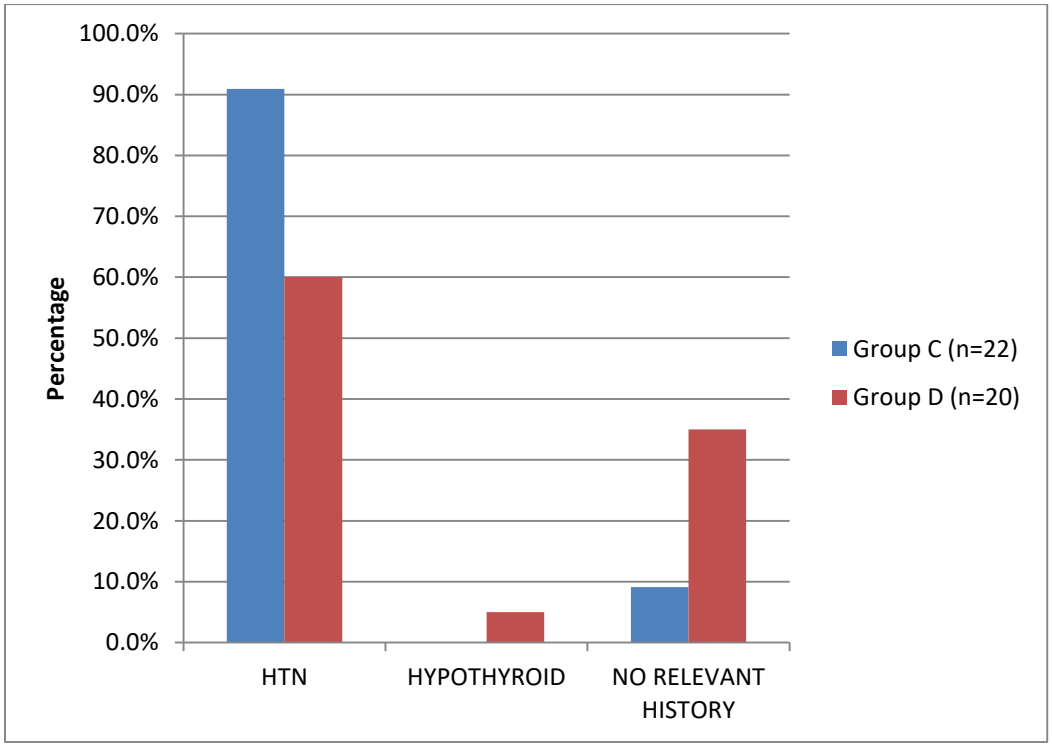



In our study in group C, 9.1% (n=2) were unbooked and 90.9% (n=20) were booked. In group D, 5% (n=1) were unbooked and 95% (n=19) were booked.

Table 9. The distribution of women who developed hypertension according to past history

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-Square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Past history | HTN | 20 | 90.9% | 12 | 60.0% | 32 | 5.517 | 0.018 |
| | Hypothyroid | 0 | 0.0% | 1 | 5.0% | 1 | | |
| | No relevant history | 2 | 9.1% | 7 | 35.0% | 9 | | |

Figure 9. The distribution of women who developed hypertension according to past history

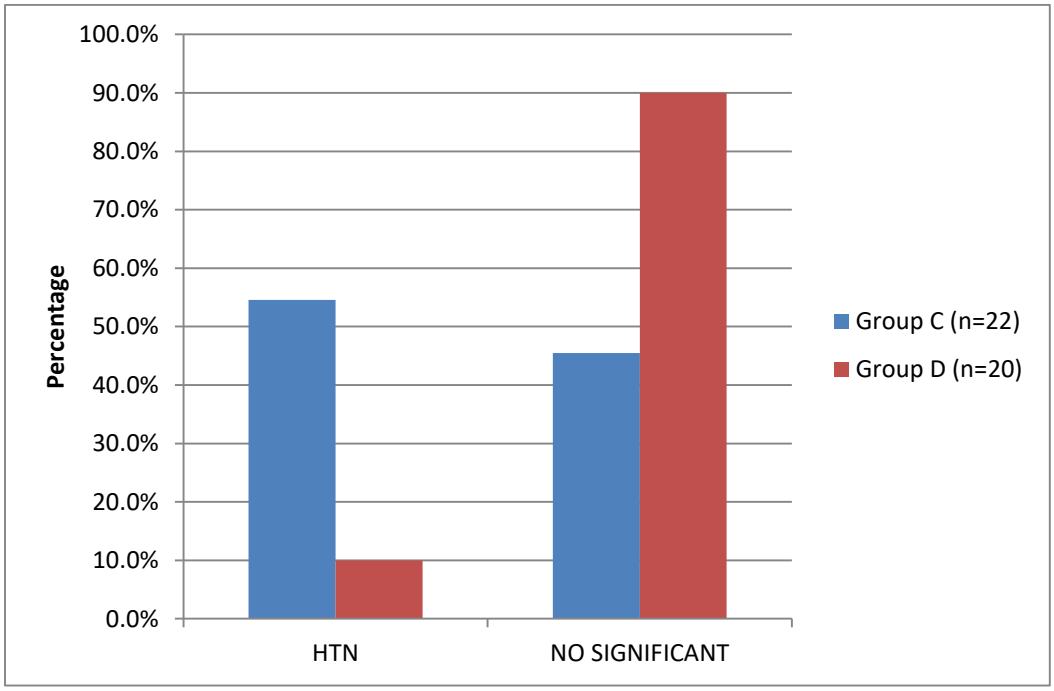



In our study the majority of women in group C, 90.9% (n=20) who had past history of hypertension in previous pregnancy, developed hypertension in this pregnancy. In group D, 60% (n=12) who had past history of hypertension in previous pregnancy developed hypertension in this pregnancy.

Table 10. The distribution of women who developed hypertension according to family history.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Family History | HTN | 12 | 54.5% | 2 | 10.0% | 14 | 6.457 | 0.011 |
| | No Significant | 10 | 45.5% | 18 | 90.0% | 28 | | |

Figure 10. The distribution of women who developed hypertension according to family history.

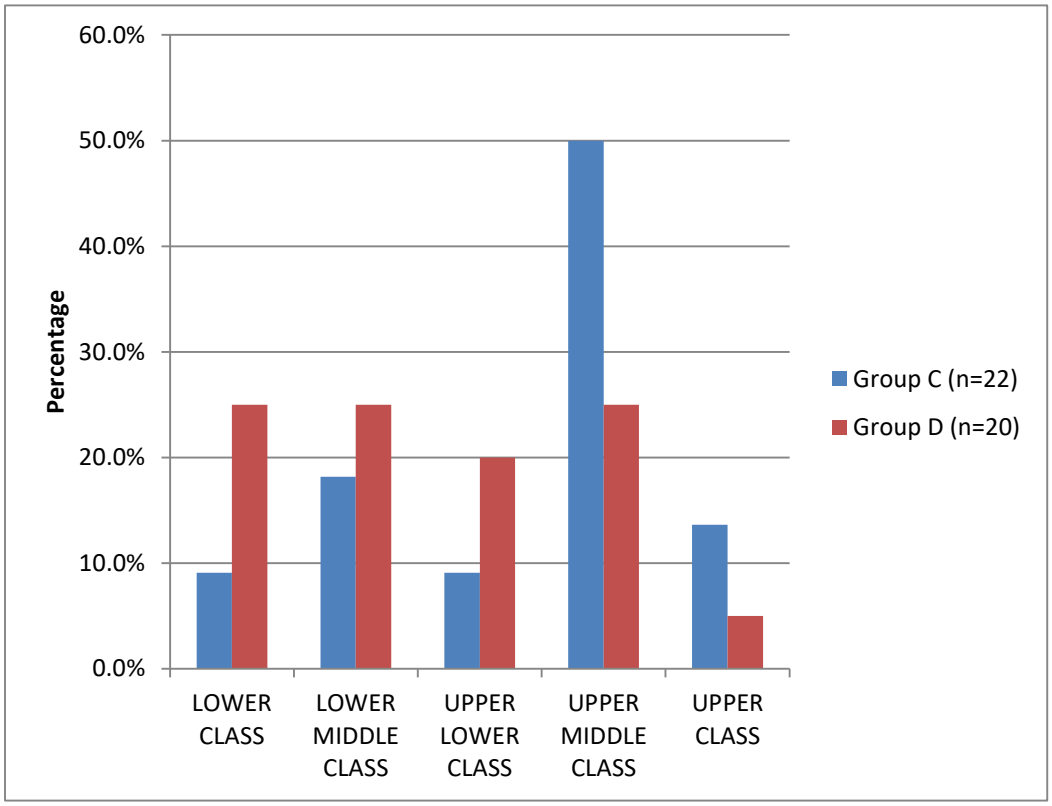



In our study, the women who developed hypertension in group C 54.5% (n=12) and in group D 10% (n=2) had significant family history of hypertension. In group C, 45.5% (10) and in group D 90% (n=18) had no significant family history.

Table 11. The distribution of women who developed hypertension according to socioeconomic status by Modified Kuppuswamy scale.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Socio economic status | Lower class | 2 | 9.1% | 5 | 25.0% | 7 | 1.909 | 0.167 |
| | Lower middle class | 4 | 18.2% | 5 | 25.0% | 9 | 0.289 | 0.591 |
| | Upper lower class | 2 | 9.1% | 4 | 20.0% | 6 | 1.018 | 0.313 |
| | Upper middle class | 11 | 50.0% | 5 | 25.0% | 16 | 2.776 | 0.096 |
| | Upper class | 3 | 13.6% | 1 | 5.0% | 4 | 0.906 | 0.341 |

Figure 11. The distribution of women who developed hypertension according to socioeconomic status by Modified Kuppuswamy scale.




In our study in group C, 9.1%(N=2) of lower class women, 18.2%(N=4) of lower middle class, 9.1%(N=2) of upper lower class ,50%(n=11) of upper middle class ,13.6%(n=3) of upper class developed hypertension.

In group D, 25%(n=5) of lower class women, 25%(n=5) of lower middle class women, 20%(n=4) of upper lower class, 25%(n=5) of upper middle class, 5% (n=1) of upper class women developed hypertension.

In our study, women in group C 50%(n=11) and group D 25%(n=5) who developed hypertension belonged to upper middle class.

B. MATERNAL PARAMETERS

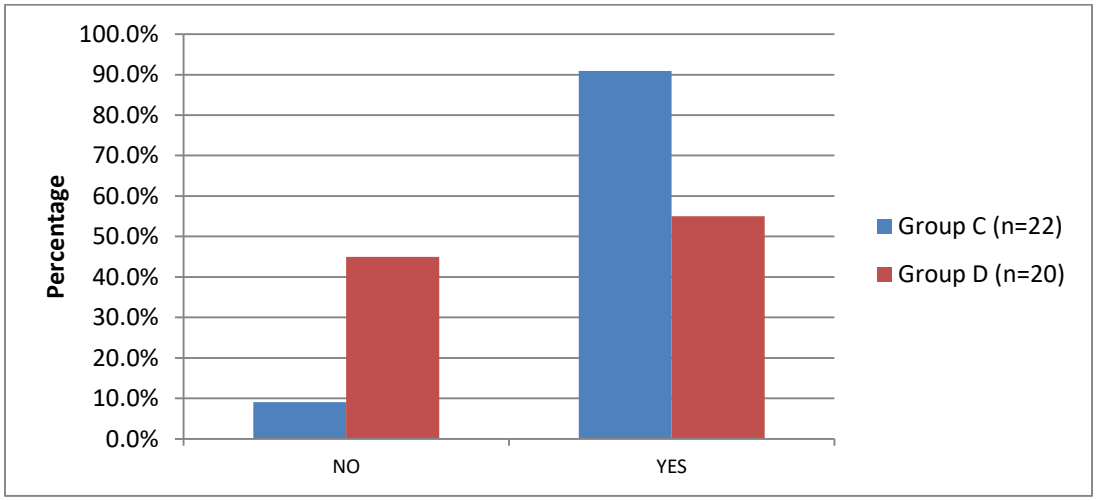
The maternal parameters under study for both groups:-

1. Symptoms-Vomiting
2. Symptoms-Headache
3. Symptoms-Blurring of vision
4. Symptoms-Epigastric pain
5. Symptoms-Pedal Edema
6. Symptoms-Generalised Edema
7. Period of gestation for development of hypertension.

Table 12. The distribution of women who developed hypertension according to development of vomiting.

| Maternal Complication | | Group C (n=22) | | Group D (n=20) | | Total | Chi-Square value | P-value |
|---|---|---|---|---|---|---|---|---|
| VOMITING | No | 2 | 9.1% | 9 | 45.0% | 11 | 6.988 | 0.008 |
| | Yes | 20 | 90.9% | 11 | 55.0% | 31 | | |

Figure 12. The distribution of women who developed hypertension according to development of vomiting.

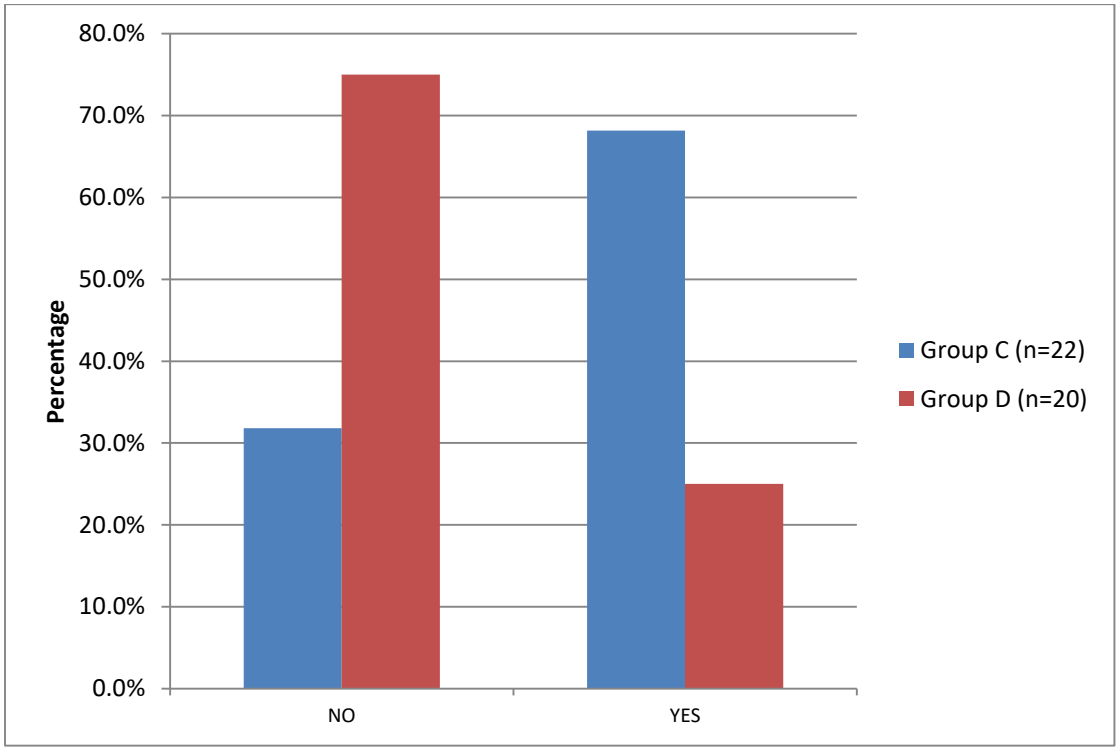



In our study the majority of women who developed hypertension in group C 90.9% (n=20) and group D 55%(n=11) had vomiting.

Table 13. The distribution of women who developed hypertension according to headache.

| Maternal complications | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Headache | NO | 7 | 31.8% | 15 | 75.0% | 22 | 7.83 | 0.005 |
| | YES | 15 | 68.2% | 5 | 25.0% | 20 | | |

Figure 13. The distribution of women who developed hypertension according to headache.

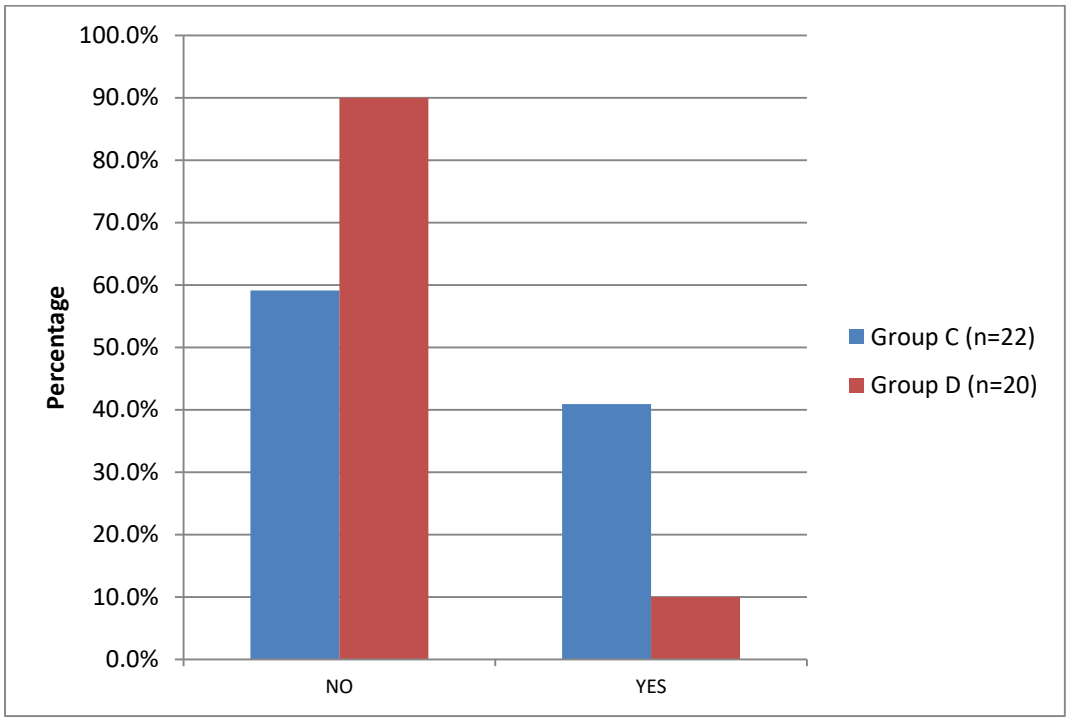



In our study, the women with lateral placenta 68.2% (n=15) with central placenta 25% (n=5) who developed hypertension had headache.

Table 14. The distribution of women who developed hypertension according to blurring of vision.

| Maternal complication | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Blurring Of Vision | No | 13 | 59.1% | 18 | 90.0% | 31 | 5.177 | 0.023 |
| | Yes | 9 | 40.9% | 2 | 10.0% | 11 | | |

Figure 14. The distribution of women who developed hypertension according to blurring of vision.

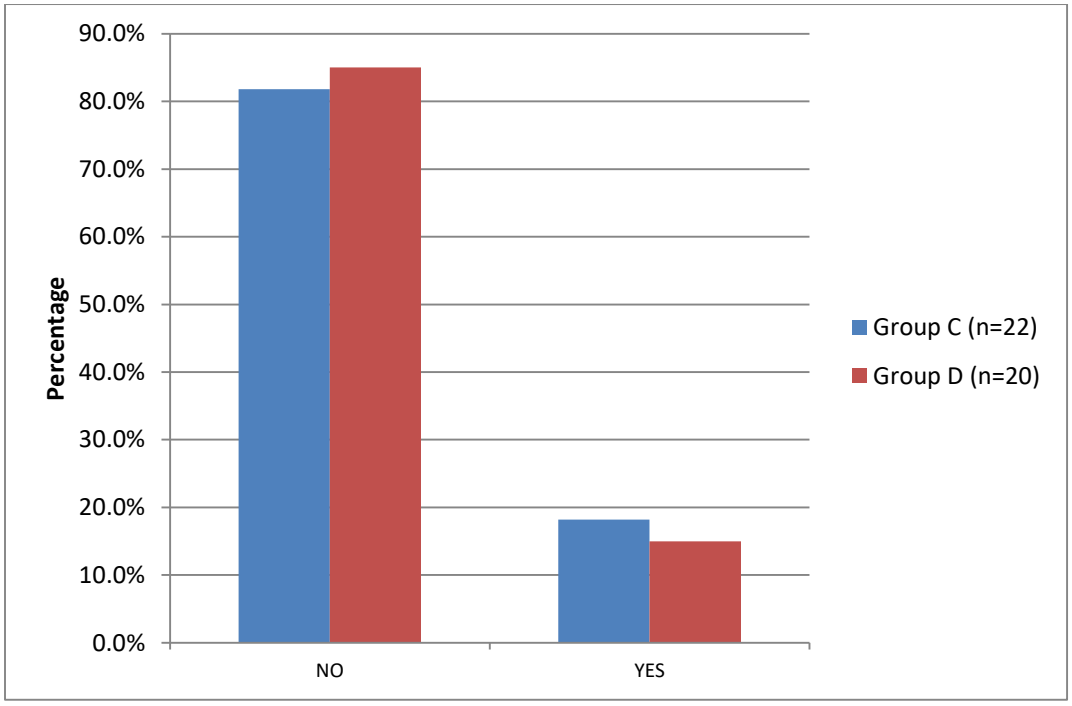



In our study, the women in group C 40.9% (n=9) and in group D 10% (n=2) who developed hypertension had blurring of vision.

Table 15. The distribution of women who developed hypertension according to epigastric pain.

| Maternal complication | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Epigastric pain | No | 18 | 81.8% | 17 | 85.0% | 35 | 0.076 | 0.782 |
| | Yes | 4 | 18.2% | 3 | 15.0% | 7 | | |

Figure 15. The distribution of women who developed hypertension according to epigastric pain.

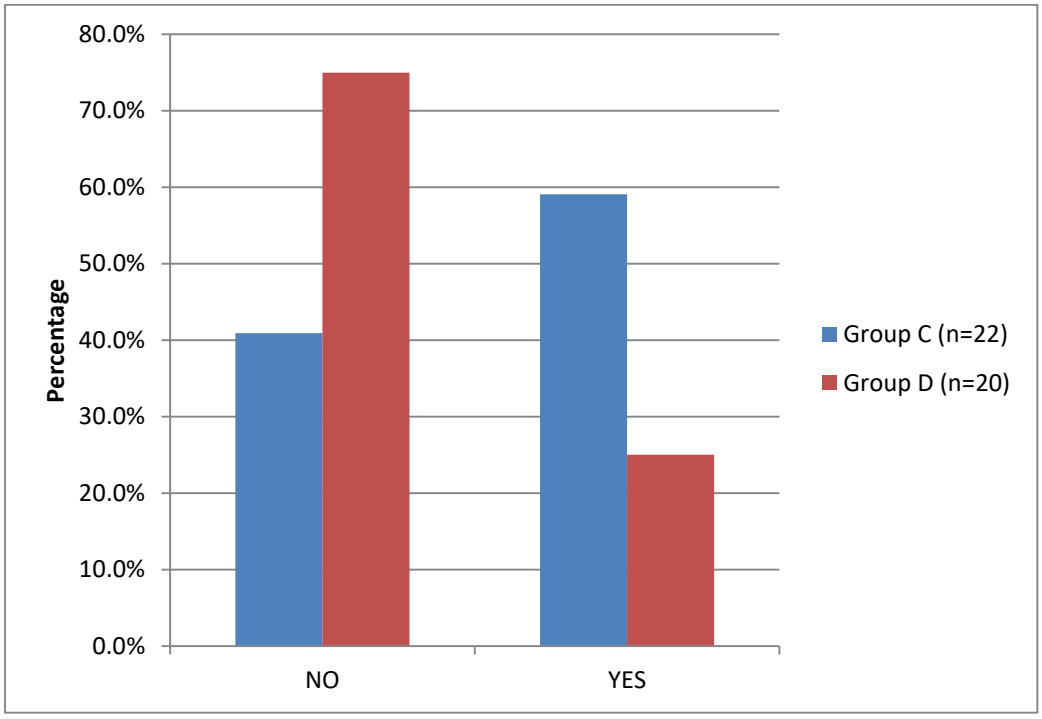



In our study in group C 18.2% (n=4) and in group D 15% (n=3) who developed hypertension had epigastric pain.

Table 16. The distribution of women who developed hypertension with/without pedal edema

| Maternal complication | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Edema_pedal | No | 9 | 40.9% | 15 | 75.0% | 24 | 4.972 | 0.026 |
| | Yes | 13 | 59.1% | 5 | 25.0% | 18 | | |

Figure 16.The distribution of women who developed hypertension with/without pedal edema

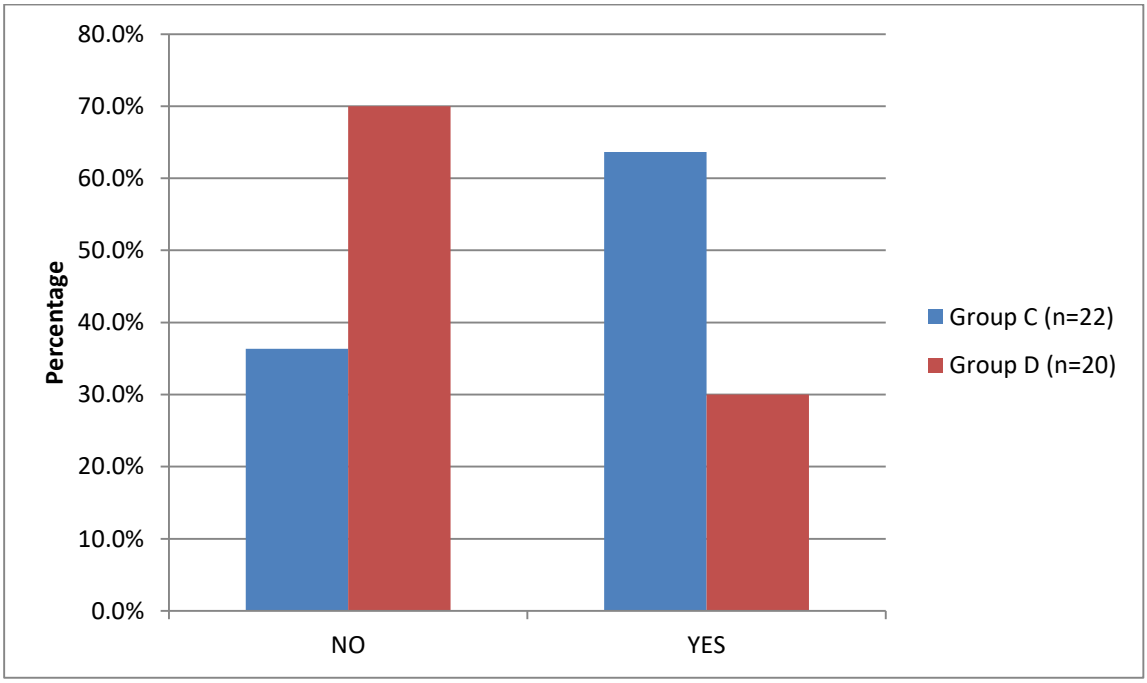



In our study, the women in group C 59.1% (n=13) had pedal edema whereas in group D 25% (n=5) women who developed hypertension had pedal edema.

Table 17.The distribution of women who developed hypertension with/without generalized edema.

| Maternal complication | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Edema_generalised | No | 8 | 36.4% | 14 | 70.0% | 35 | 4.752 | 0.037 |
| | Yes | 14 | 63.6% | 6 | 30.0% | 7 | | |

Figure 17.The distribution of women who developed hypertension with/without generalized edema.

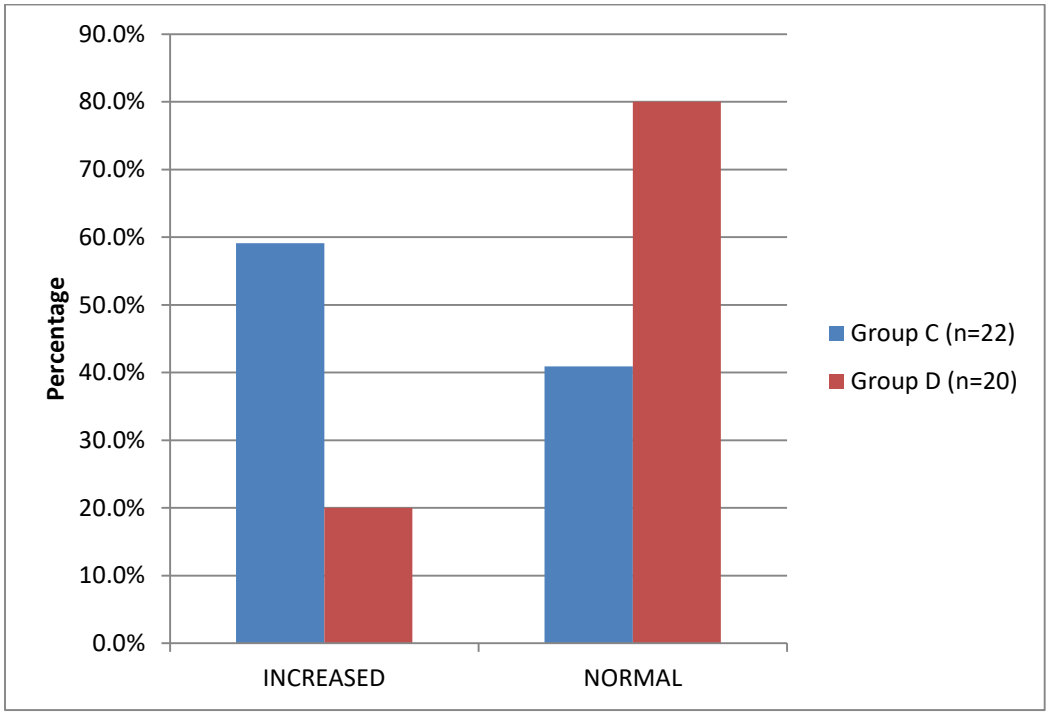



In our study, women in group C 63.6% (n=14) and in group D 30% (n=6) who developed hypertension had generalized edema.

Table 18. The distribution of women who developed hypertension according to spot Protein:Creatinine ratio

| Spot protein:creatinine ratio | | Group C (n=22) | | Group D (n=20) | | Total | Chi-squae value | P-value |
|---|---|---|---|---|---|---|---|---|
| Urine Routine | Increased | 13 | 59.1% | 4 | 20.0% | 17 | 6.669 | 0.001 |
| | Normal | 9 | 40.9% | 16 | 80.0% | 25 | | |

Figure 18. The distribution of women who developed hypertension according to spot Protein:Creatinine ratio

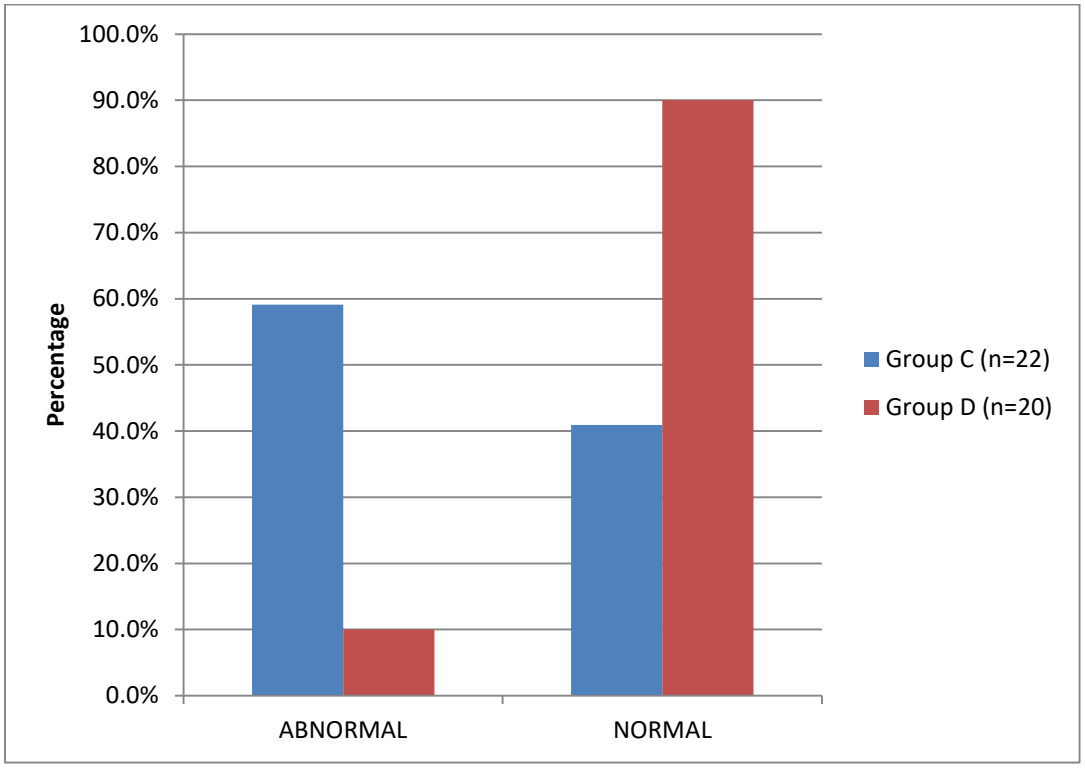



In our study, women in group C 59.1% (n=13) and in group D 20% (n=4) who developed hypertension had increased spot Protein:Creatinine ratio.

Table 19. The distribution of women according to normal/abnormal Renal Function Test

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| RFT | Abnormal | 13 | 59.1% | 2 | 10.0% | 15 | 10.996 | 0.001 |
| | Normal | 9 | 40.9% | 18 | 90.0% | 27 | | |

Figure 19. The distribution of women according to normal/abnormal Renal Function Test

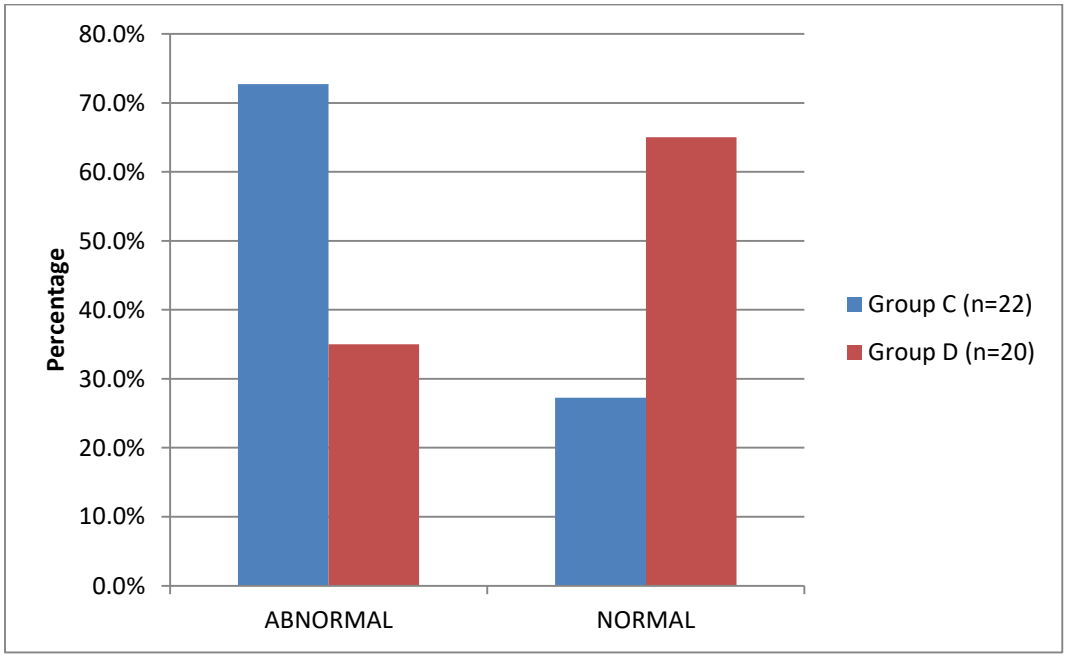



In our study, the women in group C 59.1% (n=13) and in group D 10% (n=2) who developed hypertension had abnormal Renal Function Test.

Table 20. The distribution of women according to normal/abnormal Liver Function Test

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| LFT | Abnormal | 16 | 72.7% | 7 | 35.0% | 23 | 7.644 | 0.005 |
| | Normal | 6 | 27.3% | 13 | 65.0% | 19 | | |

Figure 20. The distribution of women according to normal/abnormal Liver Function Test

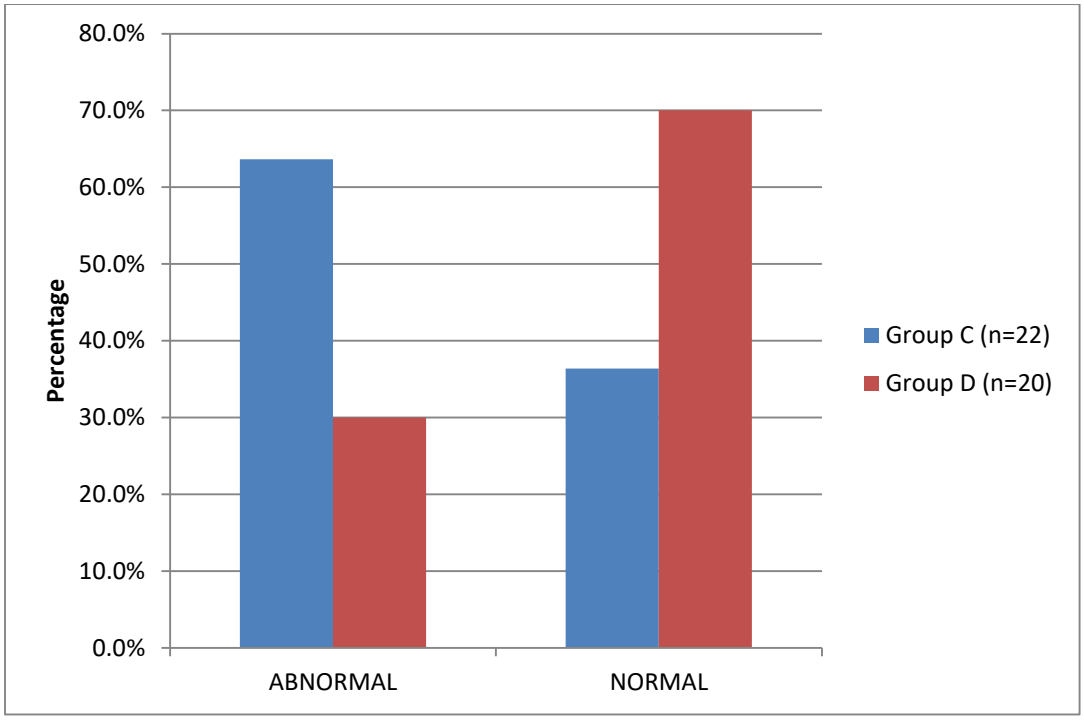



In our study the women in group C 72.7% (n=16) and group D 35%(n=7) had abnormal Liver Function Test.

Table 21.The distribution of women according to the normal/abnormal uric acid.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Uric Acid | Abnormal | 14 | 63.6% | 6 | 30.0% | 20 | 4.752 | 0.037 |
| | Normal | 8 | 36.4% | 14 | 70.0% | 22 | | |

Figure 21.The distribution of women according to the normal/abnormal uric acid.

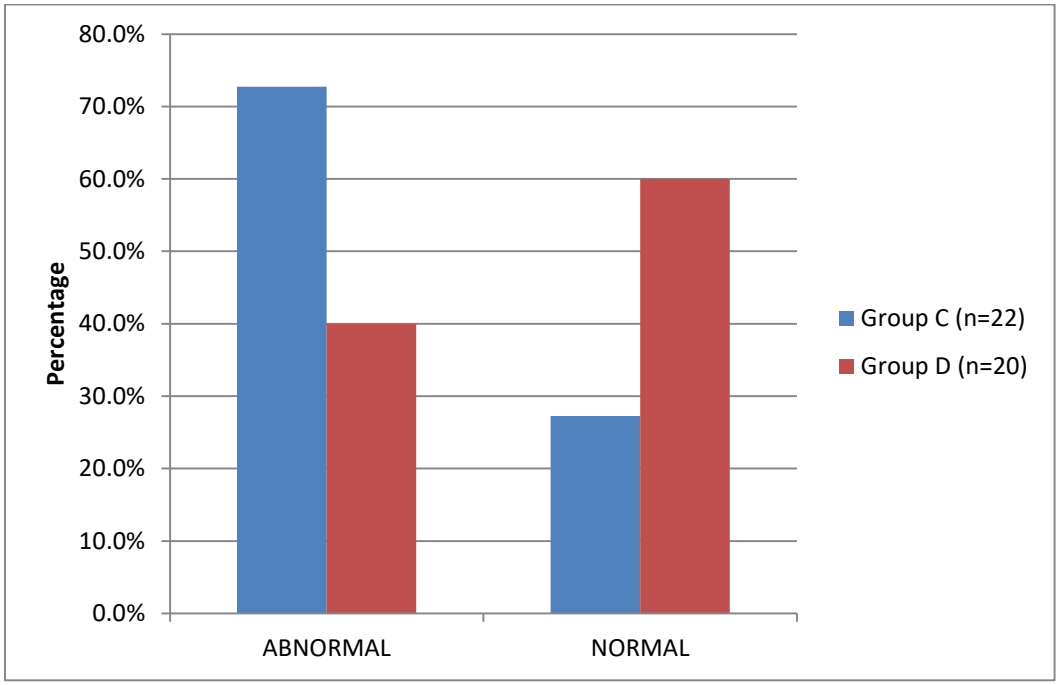



In our study the women who developed hypertension in group C 63.6% (n=14) and group D 30% (n=6) had abnormal uric acid levels.

Table 22. The distribution of women according to normal/abnormal Lactate dehydrogenase.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| LDH | Abnormal | 16 | 72.7% | 8 | 40.0% | 24 | 4.582 | 0.032 |
| | Normal | 6 | 27.3% | 12 | 60.0% | 18 | | |

Figure 22. The distribution of women according to normal/abnormal Lactate dehydrogenase.

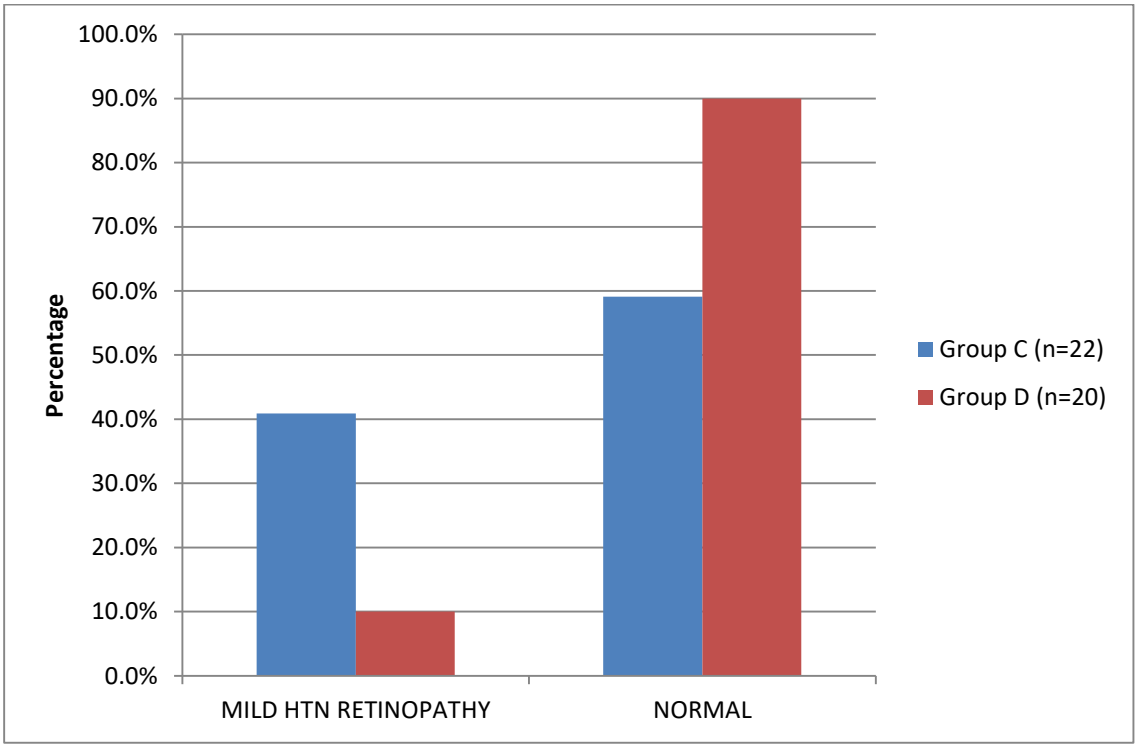



In our study the women who developed hypertension in group C 72.7% (n=16) and group D 40% (n=8) had increased Lactate dehydrogenase values.

Table 23.The distribution of women according to the normal/abnormal fundus examination.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Fundus Examination | Mild HTN retinopathy | 9 | 40.9% | 2 | 10.0% | 11 | 5.177 | 0.023 |
| | Normal | 13 | 59.1% | 18 | 90.0% | 31 | | |

Figure 23.The distribution of women according to the normal/abnormal fundus examination.

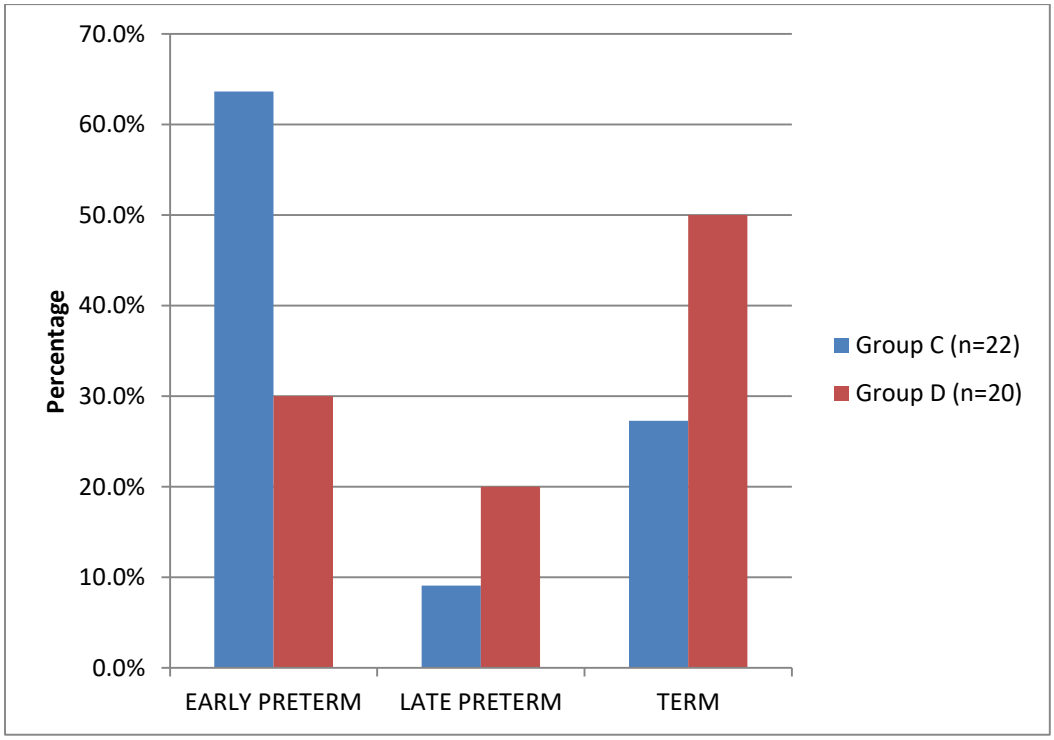



In our study, in group C 40.9%(n=9) and in group D 10%(n=2) the women who developed hypertension, had mild hypertensive retinopathy on fundus examination.

Table 24. The distribution of women according to the gestation at delivery.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Preterm / term | Early preterm | 14 | 63.6% | 6 | 30.0% | 20 | 4.752 | 0.029 |
| | Late preterm | 2 | 9.1% | 4 | 20.0% | 6 | 1.018 | 0.313 |
| | Term | 6 | 27.3% | 10 | 50.0% | 16 | 2.294 | 0.129 |

Figure 24. The distribution of women according to the gestation at delivery.

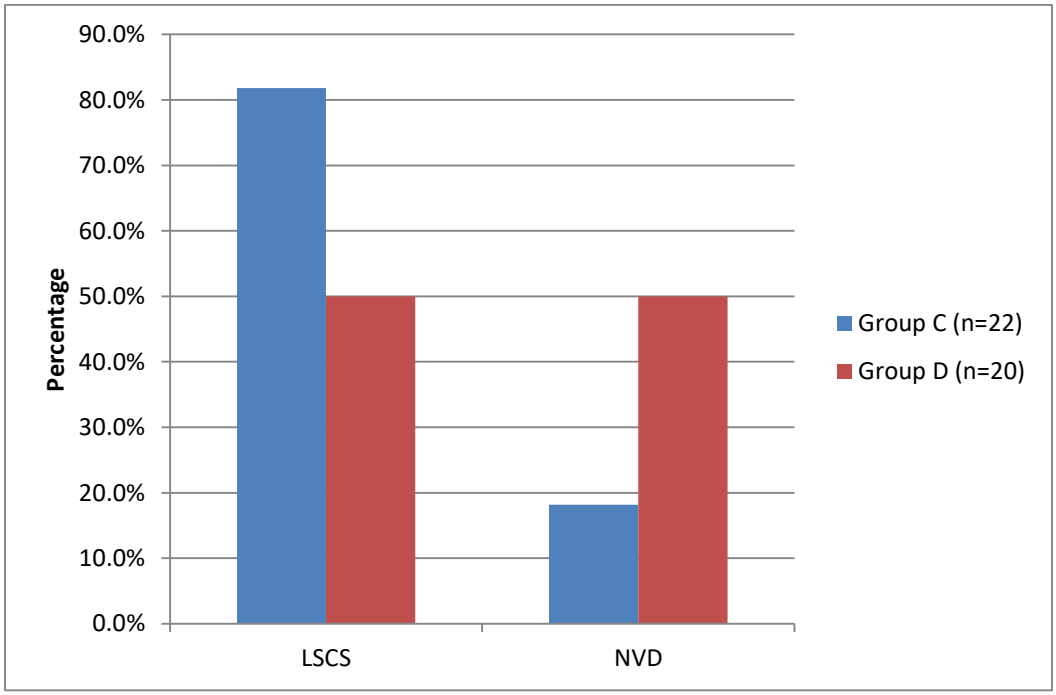



In our study, in group C 63.6% (n=14) in group D 30% (n=6) who developed hypertension had early preterm deliveries.

Table 25. The distribution of women according to the mode of delivery.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Mode Of Delivery | LSCS (Cesarean section) | 18 | 81.8% | 10 | 50.0% | 36 | 4.773 | 0.029 |
| | NVD (Normal Vaginal Delivery) | 4 | 18.2% | 10 | 50.0% | 6 | | |

Figure 25. The distribution of women according to the mode of delivery.

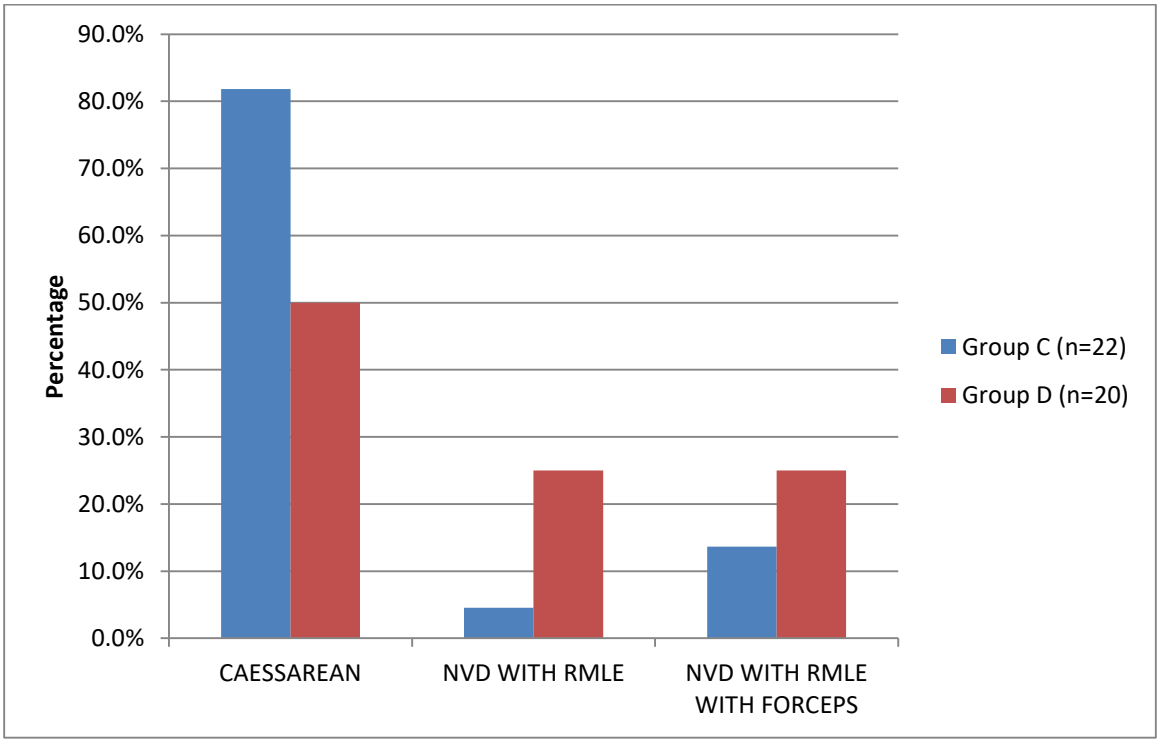



In our study, the women in group C 81.8% (n=18) and in group D 50% (n=10) who developed hypertension had cesarean section.

Table 26. The distribution of women according to the application of Forceps.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | P-value |
|---|---|---|---|---|---|---|---|---|
| Mode of delivery | Cesarean | 18 | 81.8% | 10 | 50.0% | 28 | 4.773 | 0.029 |
| | VD(VAGINAL DELIVERY) with RMLE | 1 | 4.5% | 5 | 25.0% | 6 | 3.579 | 0.058 |
| | VD(VAGINAL DELIVERY)with RMLE with FORCEPS | 3 | 13.6% | 5 | 25.0% | 8 | 0.87 | 0.348 |

Figure 26. The distribution of women according to the application of Forceps.

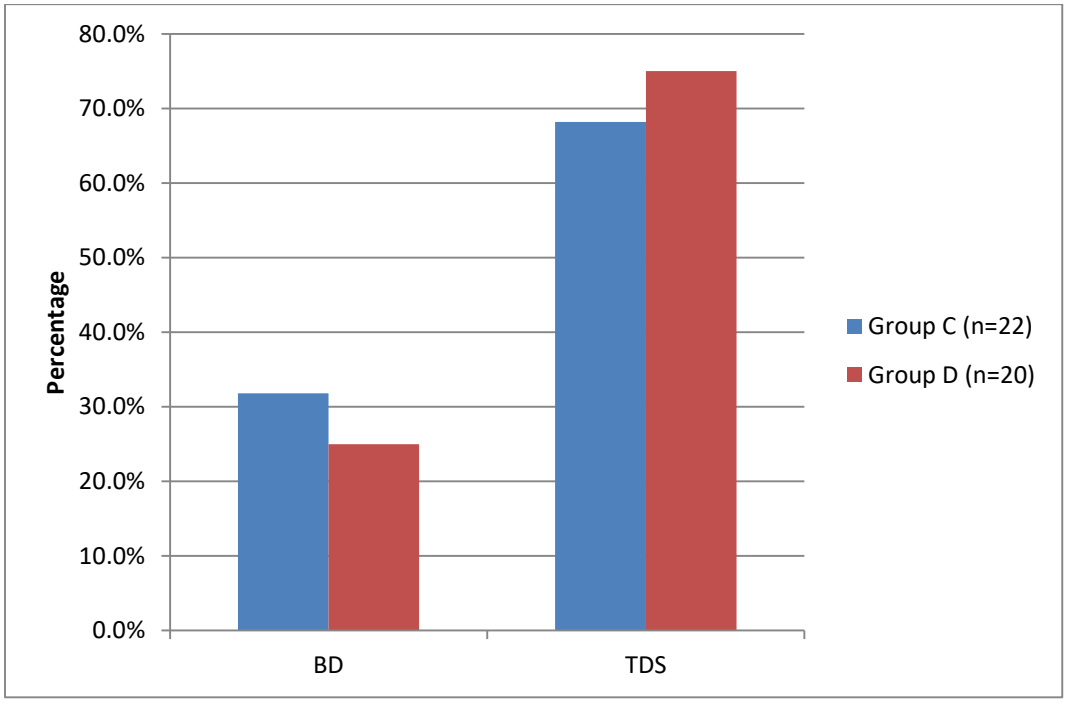



In our study the women who developed hypertension in group C 13.6% (n=3) and in group D 25% (n=5) had application of forceps.

Table 27. The frequency of women according to the antihypertensive frequency.

| | | GROUP C (n=22) | | GROUP D(n=20) | | Total | Chi-Square value | p-value |
|---|---|---|---|---|---|---|---|---|
| Antihypertensive Frequency | BD | 7 | 31.8% | 5 | 25.0% | 12 | 0.239 | 0.625 |
| | TDS | 15 | 68.2% | 15 | 75.0% | 30 | | |

Figure 27. The frequency of women according to the antihypertensive frequency.

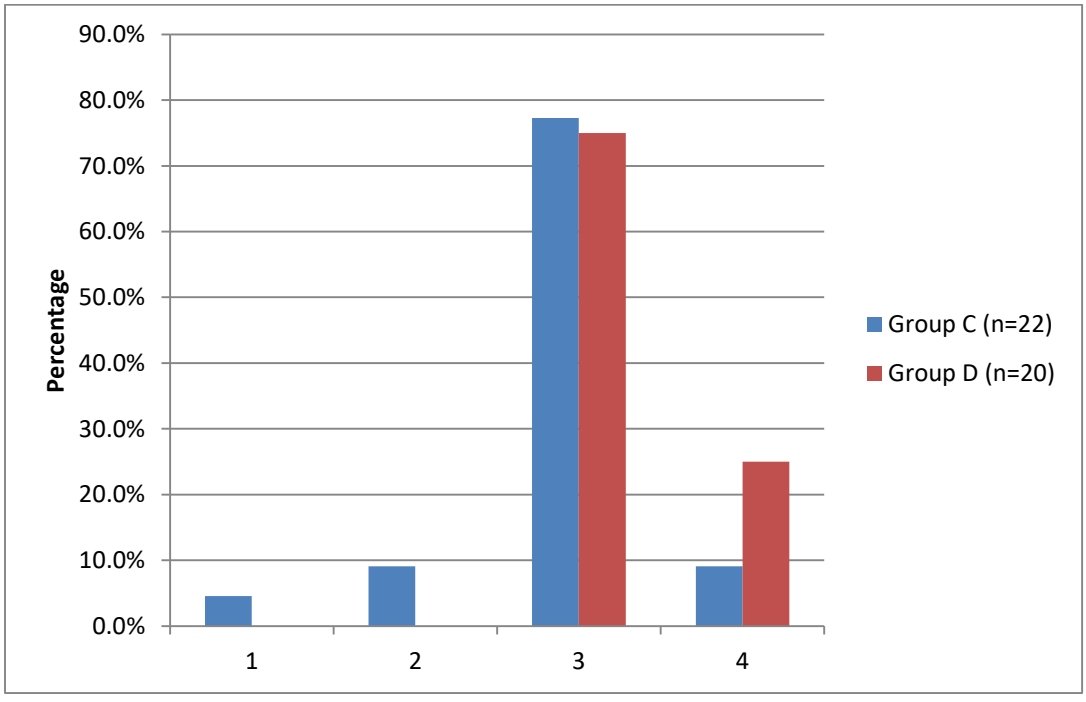



In our study, women in group C 68.2% (n=15) and in group D 75%(n=15) who developed hypertension took antihypertensive thrice daily.

Table 28. The distribution of women according to the duration of antihypertensive.

| | | GROUP C (n=22) | | GROUP D (n=20) | | Total | Chi-Square value | p-value |
|---|---|---|---|---|---|---|---|---|
| Antihypertensive Duration (Months) | 1 | 1 | 4.5% | 0 | 0.0% | 1 | 4.352 | 0.228 |
| | 2 | 2 | 9.1% | 0 | 0.0% | 2 | | |
| | 3 | 17 | 77.3% | 15 | 75.0% | 32 | | |
| | 4 | 2 | 9.1% | 5 | 25.0% | 7 | | |

Figure 28. The distribution of women according to the duration of antihypertensive.



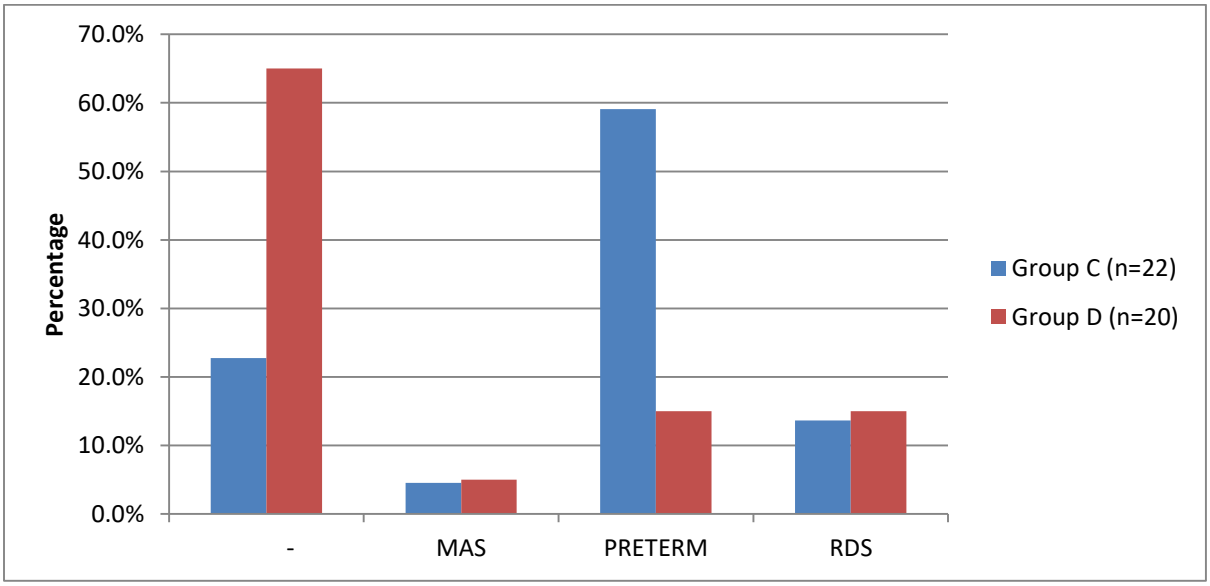

In our study the majority of women in group C 77.3%(n=17) as well as in group D 75%(n=15) took antihypertensives for 3 months.

**FETAL PARAMETERS**

Table 29. The distibution of women according to the complicating factors.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| Comorbidity | Hypoglycemia | 5 | 22.7% | 13 | 65.0% | 18 | 9.732 | 0.021 |
| | Meconium aspiration syndrome(MAS) | 1 | 4.5% | 1 | 5.0% | 2 | | |
| | Preterm | 13 | 59.1% | 3 | 15.0% | 16 | | |
| | Respiratory distress syndrome (RDS) | 3 | 13.6% | 3 | 15.0% | 6 | | |

Figure 29. The distibution of women according to the complicating factors.



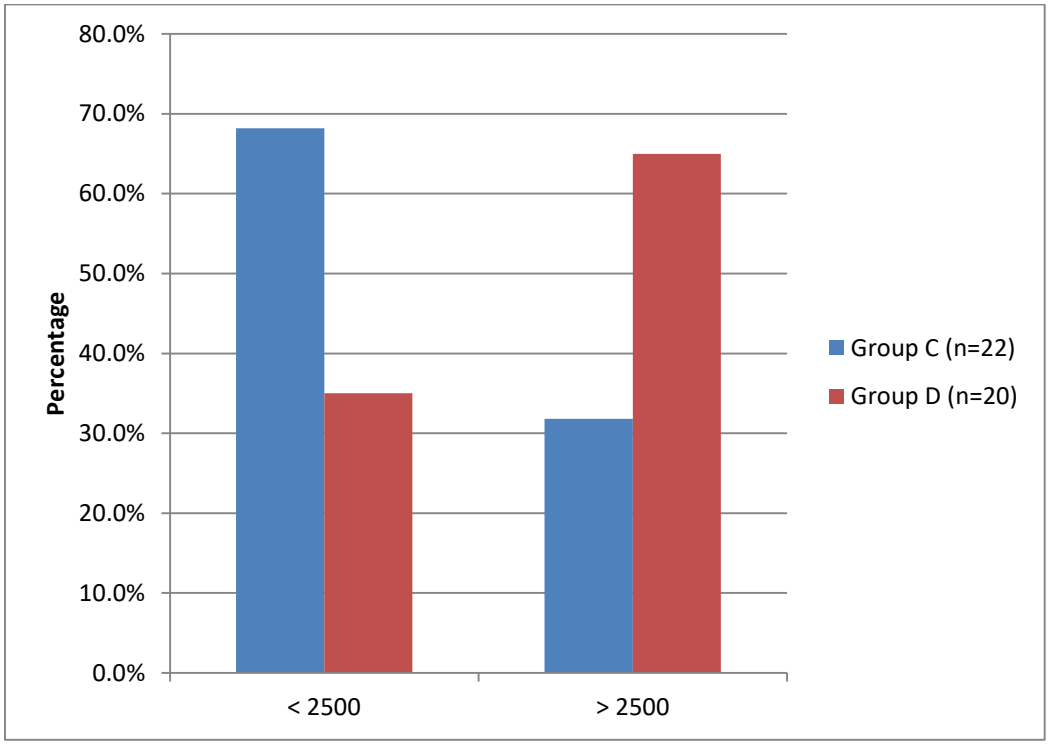

In our study in group C,22.7%(n=5) had hypoglycemia, 4.5%(n=1) had meconium aspiration syndrome, 59.1% (n=13) were preterm, and 13.6%(n=3) had respiratory distress syndrome .

In group D,65%(n=13) had hypoglycemia, 5%(n=1) had meconium aspiration syndrome 15%(n=3) were preterm, 15%(n=3) had respiratory distress syndrome.

Table 30.The distribution of women according to the birth weight.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| Weight group | < 2500 | 15 | 68.2% | 7 | 35.0% | 22 | 4.624 | 0.031 |
| | > 2500 | 7 | 31.8% | 13 | 65.0% | 20 | | |

Figure 30.The distribution of women according to the birth weight.

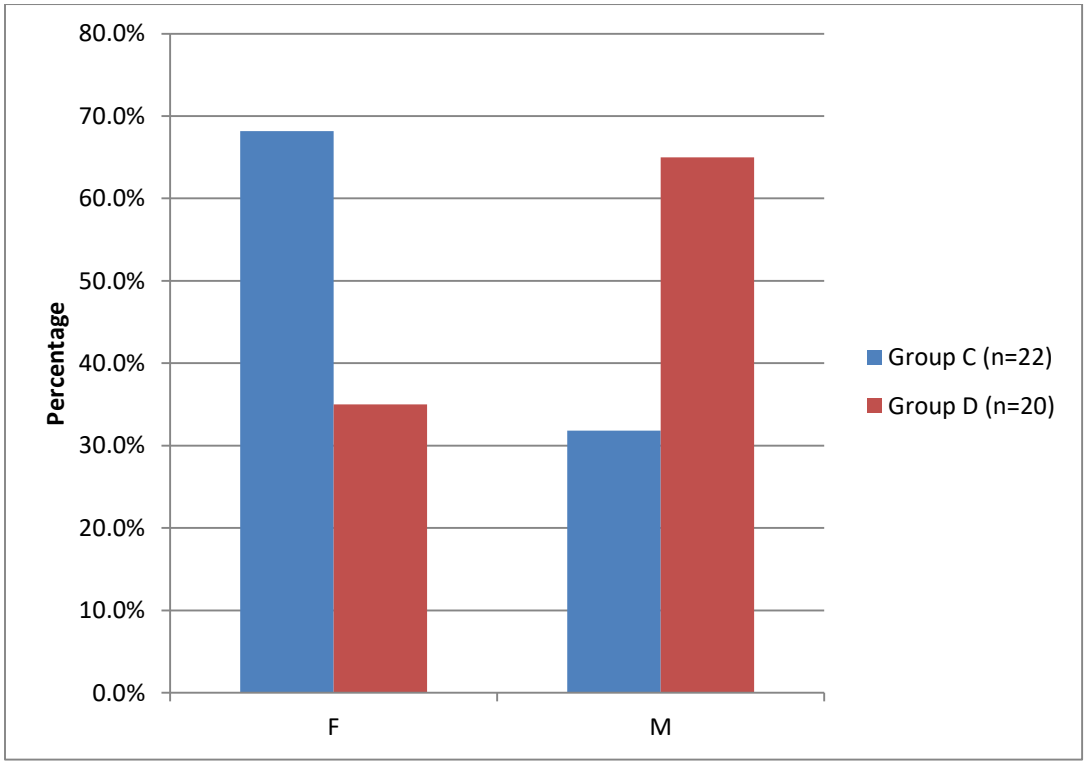



In our study, in group C 68.2% (n=15) and in group D, 35% (n=7) neonates were low birth weight.

Table 31. The distribution of women according to sex of newborn.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| Sex | F | 15 | 68.2% | 7 | 35.0% | 22 | 4.624 | 0.031 |
| | M | 7 | 31.8% | 13 | 65.0% | 20 | | |

Figure 31. The distribution of women according to sex of newborn.

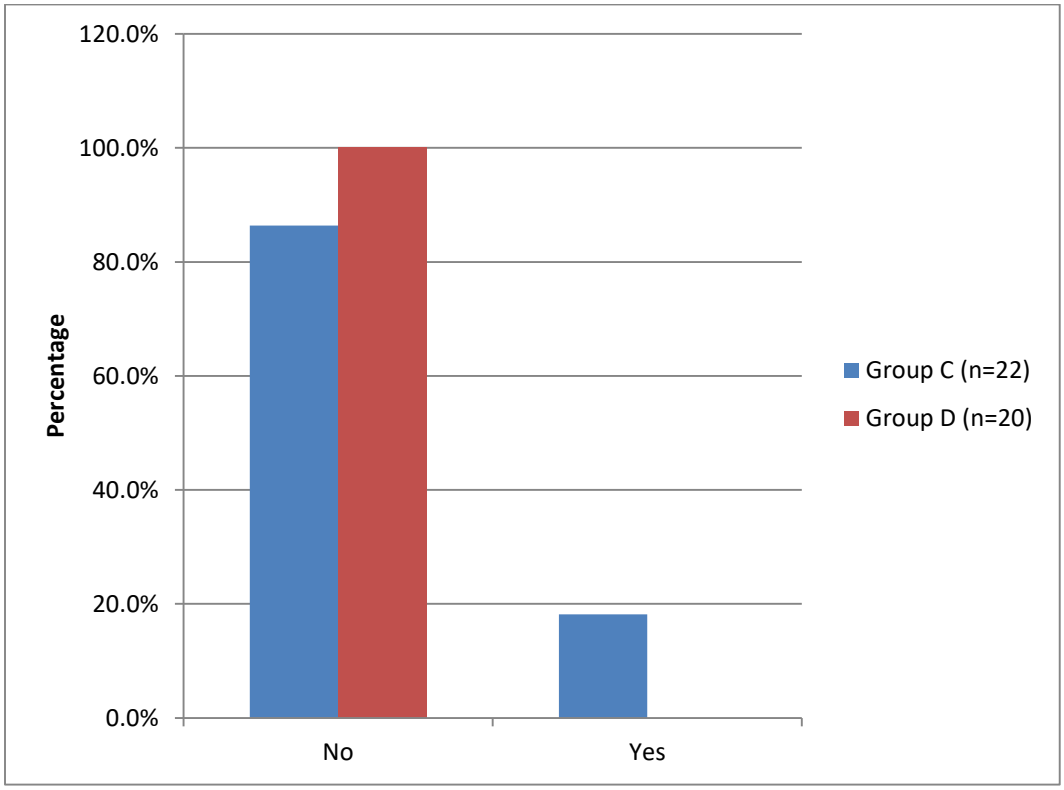



In our study in group C 68.2% (n=15) were female ,31.8%(n=7) were male and in group D 35% (n=7) were female ,65% (n=13) were male.

Table 32. The distribution of women according to the neonatal death.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| Neonatal Death | No | 19 | 86.4% | 20 | 100.0% | 39 | 4.019 | 0.045 |
| | Yes | 4 | 18.2% | 0 | 0.0% | 4 | | |

Figure 32. The distribution of women according to the neonatal death.



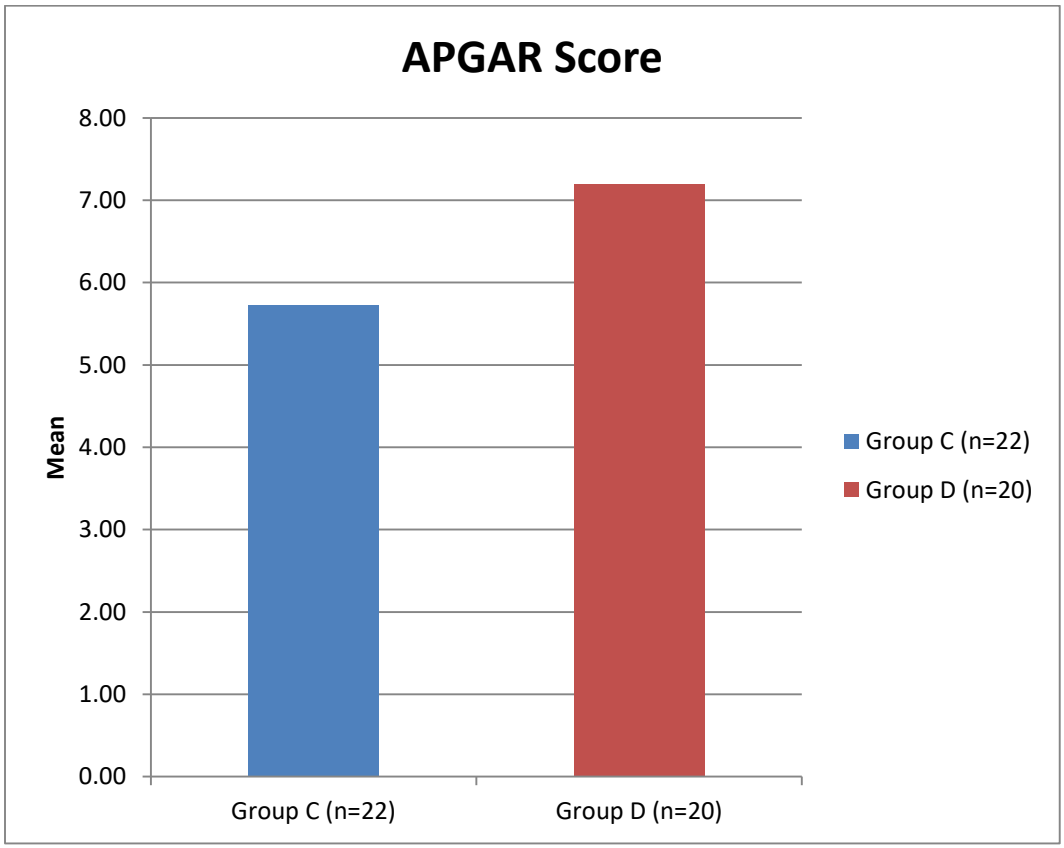

In our study, in group C, 18.2% (n=4) neonatal death was present and no neonatal death in group D.

Table 33. The distribution of women acccording to the APGAR score.

(Appearance, Pulse, Grimace, Activity and Respiration)

| | Group C (n=22) | | Group D (n=20) | | t | p-value |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| APGAR Score | 5.73 | 1.35 | 7.20 | 1.79 | 3.021 | 0.004 |

Figure 33. The distribution of women acccording to the APGAR score. (Appearance, Pulse, Grimace, Activity and Respiration)



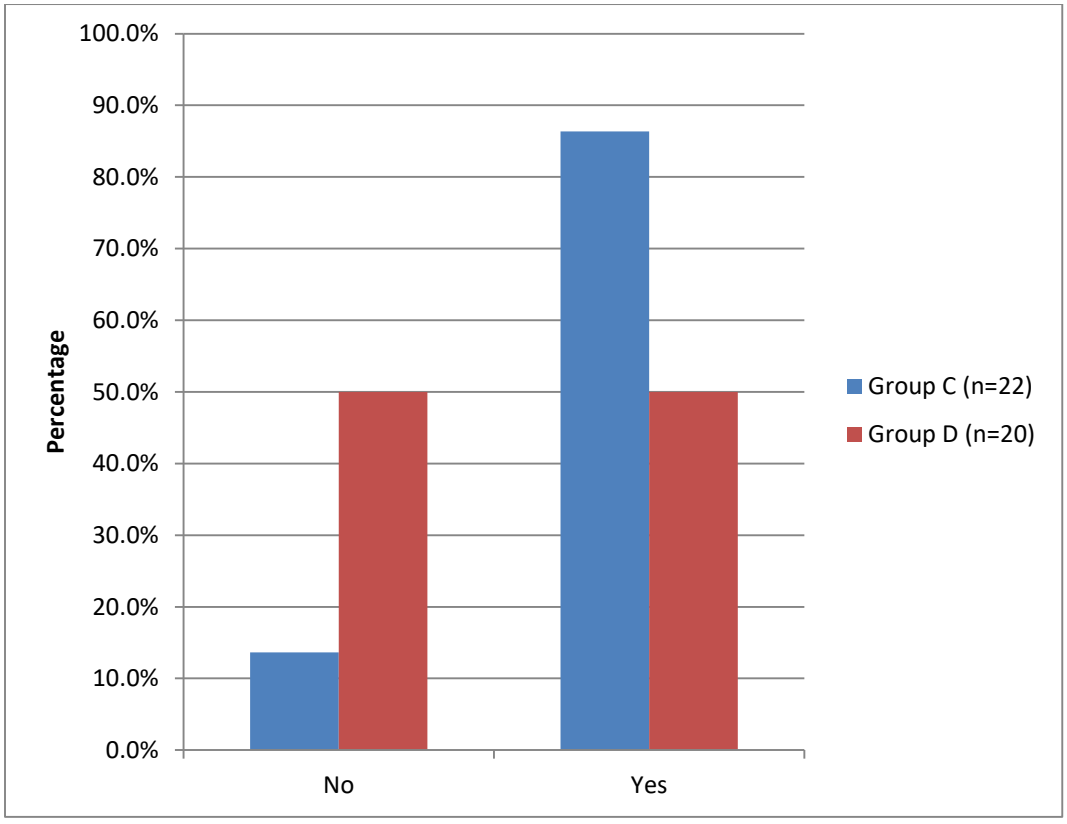

In our study in group C, mean APGAR score at 1 minute was 5.73 and mean APGAR score in group D was 7.20. The mean APGAR score in group C was lower than that in group D.

Table 34. The distribution of women according to the NICU

(Neonatal Intensive Care Unit) admission.

| | | Group C (n=22) | | Group D (n=20) | | Total | Chi-square value | p-value |
|---|---|---|---|---|---|---|---|---|
| NICU Admission | No | 3 | 13.6% | 10 | 50.0% | 13 | 6.4825 | 0.011 |
| | Yes | 19 | 86.4% | 10 | 50.0% | 29 | | |

Figure 34. The distribution of women according to the NICU (Neonatal Intensive Care Unit) admission.




In our study in group C, 86.4% (n=19) and in group D 50% (n=10) neonates had NICU admissions.

## DISCUSSION

Hypertensive disorder is the most common medical condition complicating pregnancy. It complicates upto 7-10% of pregnancies of which Preeclampsia/Eclampsia contributes 70% and chronic hypertension 30%[1]. It is said that pre-eclampsia, eclampsia contribute to death of a woman every three minutes [2].The overall risk of preeclampsia in laterally situated placenta is greatly increased due to the inadequate blood supply[2].

In our study , total women enrolled were 100.

Group A-Women with lateral placenta.

Group B- Women with central placenta.

Group C- Women with lateral placenta who developed hypertension.

Group D- Women with central placenta who developed hypertension.

**DEMOGRAPHIC PARAMETERS**

In our study, 45.5% (n=10) women in group C and 50% (n=10) women in group D who developed hypertension belonged to age between 26-30 years. The hypertension during pregnancy.is common in extremes of age of reproductive years. In our study, the age distribution in both the groups C and D was similar. In a similar study conducted by Aggarwal et al., in 2014, who studied the relation between placental laterality in second trimester ultrasound and development of preeclampsia at term, 43.6% of pregnant women were from age 21-25 years, and had laterally located placenta on ultrasound done at 18-24 weeks, who developed preeclampsia[55].

In our study, 50% (n=11) in group C and 40% (n=19) in group D, women were overweight. The p value is 0.516 which shows that there is no statistically significant relationship between BMI and hypertensive diseases of pregnancy in our study. In contrast, a study done by Corrie MacDonald -Wallis et al. in 2013, which was to study gestational weight gain as a risk factor for hypertensive disorders of pregnancy, on 12,522 women it was observed that greater pre-pregnancy weight was associated with an increased risk of gestational HTN and preeclampsia[56]. One of the independent risk factors of hypertension in pregnancy is increased body fat

mass that is reflected by overweight status.[57]. Fat cells have been characterised to be sensitive to lipolysis and their aptitude to produce high quantities of inflammatory cytokines. This inflammatory response is found to participate in the blood pressure elevation and end organ damage[57]. Increased adipose tissue, release a variety of adipokines that are related to decrease in production of nitric oxide that has been associated with the endothelial dysfunction and arterial hypertension[57]. However in our study, due to small sample size significant association between BMI and hypertension could not be found.

In our study, 86.4% (n=19) in group C and 55% (n=11) women in group D, who developed hypertension had urban background. The p value is 0.024 which is statistically significant. In a similar study conducted by Deborah van Middendorp et al., in 2013, who studied the rural and urban differences in blood pressure and pregnancy induced hypertension amongst pregnant women. The study was done on 967 pregnant women out of which 677 were rural and 290 urban pregnant women. The women in urban region had higher mean BP than women in rural region ($p<0.001$)[58]. The difference between the urban and rural region was explained by difference in their BMI. Overweight and obesity are increasing problems in cities which lead to increased free fatty acid levels and relative insulin resistance. If left unchecked, the increasing prevelance of overweight and obesity will exaberate pregnancy induced hypertension[58].

Education exerts influence through lifestyle and dietary habits. It has been found that educational attainment had powerful influence on the level of blood pressure. In our study, 68.2% (n=15) in group C and 50% (n=10) in group D, women were postgraduates. The p value is 0.231 which is not statistically significant. This has been supported by a study conducted by Ramesh K et al., in 2014, for evaluating risk factors for preeclampsia at a tertiary care hospital in Karnataka in the year 2014, who found that 22% of cases were illiterate whereas 20% of the controls were illiterate (p-0.68) which was statistically insignificant[59]. In contrast, a study by Ambika Rai et

al., in 2020, on lateral placenta as a predictor for development of preeclampsia, it was concluded that majority women (38.7%) were educated up-to higher secondary and 27.4% were postgraduates[43].

. In our study, 68.2% (n=15) women in group C and 35% (n=7) women in group D, were homemaker. The p value is 0.031 which is statistically significant. In a study conducted by Jaap Jan Nugteren et al., in 2012, on work related maternal risk factors, the risk of Pregnancy induced hypertension and Preeclampsia during pregnancy on 4465 pregnant women, no association was found between any of the work related risk factors such as prolonged standing or walking, heavy lifting, night shifts, working hours, with hypertensive disorders of pregnancy[60]. Homemakers in this part of the world are exposed to more stress due to heavy physical activity which they are supposed to do on day to day basis during pregnancy thus increasing chances of developing hypertensive disease of pregnancy. In our study, 81.8% (n=18) in group C and 50% (n=10) in group D, women were primigravida. The p value is statistically significant (0.029). Preeclampsia mostly affects the first pregnancy as these women are exposed to chorionic villi for the first time. Nulliparity is a well-known risk factor for hypertensive disease of pregnancy. Nulliparous women have 2-3 times increased risk of developing hypertension in pregnancy. Higher circulating sFlt1 levels and sFlt1/PIGF ratios in primigravida patients than in multiparous pregnancies suggests an association with angiogenic imbalance proving that nulliparity is a risk factor for the development of preeclampsia as per the study of Bdolah Y et al., in 2014, which was on nulliparous women to study the relationship between nulliparity and preeclampsia which may be explained by altered circulating soluble fms-like tyrosine kinase 1[61]. In a similar study done by Ramya Kakku et al., in 2017, to study the lateral location of placenta on ultrasound as a predictive test for preeclampsia, found that preeclampsia is more common in primigravida (72%) than in multigravida (28%) which was same as in our study[1]

In our study, 90.9% (n=20) in group C and 95% (n=19) in group D, women were booked. The women who were followed for development of hypertension in our study were mostly booked. The p value is 0.607 which is not statistically significant. As per the government of India norms every pregnant women should have four antenatal checkups. WHO has increased number of contacts from four to eight. Hypertensive disorder of pregnancy is significantly high in women having poor access to antenatal care. Registering for skilled antenatal supervision within appropriate health facility remains a great challenge in low resource setting countries like India so there is low detection of hypertensive disorders of pregnancy among unbooked and higher detection among booked. It is found that Pregnancy induced hypertension is not totally a preventable disease but maternal and fetal complications due to hypertensive disease of pregnancy can be halted at mild stage by quality antenatal care resulting in good outcome. In study conducted by P Jayasree et al., in 2016, a comparative study of maternal outcome in booked and unbooked pregnancy induced hypertension in mothers in tertiary care hospital, it was found that severe pregnancy induced hypertension and complications were more in unbooked cases than the booked cases and this was statistically significant $p<0.05$[62].

In our study, 90.9% (n=20) in group C and 60% (n=12) in group D, women had significant past medical history of hypertension .The p value is statistically significant (0.018). In our study, it was found that women who had past history of hypertension in previous pregnancy had greater risk of hypertensive disorder of pregnancy .Genetic factors have been clearly found to contribute to the susceptibility to preeclampsia[63]. In a similar study by Ufaque Muzaffar et al.,in 2020 to study Early predictors of Preclampsia: A Prospective Screening Study, it was found that women who had past history of hypertension(10%) were significantly associated with preeclampsia[64].

In our study, 54.5% (n=12) in group C and 10% (n=2) in group D, women had family history of hypertension. The p value is 0.011 which is statistically

significant. In a study by Mulualem Endeshaw et al., in 2016, on 453 pregnant women to study the family history of hypertension, increases the risk of preeclampsia in pregnant women, it was found that family history(AOR=11.16;95% CI-5.41-41.43) was a dominant risk factor for development of preeclampsia in pregnant women[63]. A family history (mother, sister or both) of preeclampsia is associated with a fourfold increased risk of preeclampsia[63].

Socioeconomic status is an important factor which determines access to health care including family planning, precocious marriage and development of hypertensive disease of pregnancy. In our study, 50% (n=11) in group C and 25% (n=5) in group D, women belonged to upper middle class. The p value is not statistically significant (0.096). In high socioeconomic strata unhealthy nutritional habits explain the increased prevelance of preeclampsia .In a similar study by Ambika Rai et al., in 2020, on lateral placenta as a predictor for development of preeclampsia, it was found that most of the women with preeclampsia hailed from middle class (55.7%) followed by low (22.6%), lastly high class(21.7%)[43]. In a similar study by Ananya Priyadarshini et al., done in 2019 on 100 cases it was observed that preeclampsia was more common in middle class 81% followed by lower 18% and lastly upper class 1%[53].

MATERNAL PARAMETERS

VOMITINGS

In our study, 90.9% (n=20) in group C and 55% (n=11) in group D, women had vomiting as a symptom. The p value for determining significance of vomiting as a severe feature is 0.008. Data shows a statistical significant association between vomitings as a severe feature and preeclampsia. Abnormal levels of both relaxin, human chorionic gonadotropin (HCG) have been implicated with gastrointestinal motility and gastric dysrhythmias contributing to nausea and vomiting[65]. Vomiting can lead to dyselectrolytemia, renal abnormalities, liver dysfunction, raised haematocrit leading to hypertensive disorder of pregnancy. In a similar study, conducted

by Arthur Chortatos et al., in 2015 which was on pregnancy complications and birth outcomes among women experiencing nausea only or nausea and vomiting during pregnancy on 51675 women it was found that women with nausea, vomiting (33%) had greater rates of high blood pressure during pregnancy, proteinuria and preeclampsia than symptom free group (28%)[66].

In our study, 68.2% (n=15) in group C and 25% (n=5) in group D, women had headache as a symptom. The p-value is 0.005 showing significant relationship between headache and preeclampsia. In a similar study by Facchinetti et al., in 2005, which was done to study the relationship between headache and preeclampsia,, headache was significantly associated with preeclampsia (47/75) as compared to controls(19/75) ,it was concluded that pregnancy-induced hypertension (pre-eclampsia) is linked to increased vascular reactivity, increased vasoconstrictors, endothelial damage and platelet hyperaggregation causing headache[67].

The complications of preeclampsia have been found to involve multiple systems and organs, the eye and visual system are of no exception. Visual symptoms have been found to concern up to 25% of patients with severe preeclampsia and 50% of patients with eclampsia[68]. Various ocular manifestations are found in preeclampsia/eclampsia. The most common visual complaint is Blurred vision. Retinal vasospasm and resistance to blood flow have been found to be possible explanation for visual symptoms. Most common ocular findings in preeclampsia is focal/generalised arteriolar narrowing[68]. In our study, 40.9% (n=9) in group C and 10% (n=2) in group D women had blurring of vision as a symptom. The p value for determining significance of blurring of vision as a severe feature is 0.023 that is significant which suggests that blurring of vision can predict development of preeclampsia. In a similar study conducted by Tadin et al., in 2001 to study Hypertensive retinopathy and preeclampsia on 40 women, the degree of hypertensive retinopathy was found to be directly proportional to the severity of preeclampsia and significant correlation was found between

them (p-0.033). It was found that degree of hypertensive retinopathy in women with preeclampsia is valid and reliable prognostic tool to determine the severity of preeclampsia[69].

It has been observed that 10% of pregnant women with severe preeclampsia have liver enzyme abnormalities[70]. The resulting pain (epigastric or right upper quadrant abdominal pain) is usually associated with elevated serum hepatic transaminase levels. The hemorrhage in the liver may extend beneath the hepatic (Glison's) capsule causing it's stretching and thus the pain. There is 2-3 fold increase in alanine and aspartate aminotransferases[70]. In our study, 18.2% (n=4) in group C and 15% (n=3) women in group D had epigastric pain as a symptom for severe feature. However, in our study, the p value for determining significance of epigastric pain as a symptom for severe feature is 0.782 which does not show a statistically significant relationship suggesting that epigastric pain cannot predict development of preeclampsia. Arturo Juarez- Azpilcueta et is al., in 2003, found that hepatic hemorrhage occurs rarely, specific symptoms could be identified through identification of triad -epigastric pain, hypotension without evident bleeding cause and clinical data of hypertensive disease of pregnancy[71].

In our study, 59.1% (n=13) in group C and 25% (n=5) in group D had pedal edema. The p value is 0.026, which shows that there is significant relation between pedal edema and hypertensive disorder of pregnancy. In a study by S Ananth Karumanchi et al it., in 2005, to study perspective in renal medicine: preeclampsia, it was found that Preeclampsia is associated with a characteristic glomerular lesion, ''glomerular capillary endotheliosis'' - glomerular capillary lumen is enlarged due to endothelial and mesangial swelling and hypertrophy leading to proteinuria and thus edema[65].

In our study, 63.6%(n=14) in group C and 30%(n=6) in group D, women had generalized edema. The p value is 0.037 which shows that there is significant relation between the generalised edema and hypertensive disorder of pregnancy. Decreased GFR, increased capillary permeability

and hypoalbuminemia might contribute to the edema formation[65]. In a similar study by Rajiv Aggarwal et al., in 2008, it was concluded that detection of edema is of substantial importance and its presence is independently linked to systolic hypertension[72].

In our study, 59.1% (n=13) in group C and 20%(n=4) in group D, women had increased spot P:C ratio. The p value is 0.001 which shows that there is significant relationship between spot P:C ratio and hypertensive disorder of pregnancy. The glomerular enlargement, deposition of IgM fibrin, thickened epithelial tuft, swelling of mesangial cells, moderate or gross ballooning of loops cause proteinuria resulting in increased urinary spot P:C ratio. In a similar study conducted by Oya Demirci et al.,in 2015, which was done to study spot P:C ratio as an alternative for 24-hour urine protein, it was found that spot protein/creatinine ratio can be used as a good predictor(CI:0.94-0.99) for remarkable proteinuria and it could be used as a rapid alternative test in preeclamptic patients not to delay implementation of treatment[73].

In our study, 72.7%(n=16) in Group C and 35%(n=7) in group D, women had abnormal LFT. The p value is 0.005 which shows that there is significant relationship. The hemorrhages in liver and hepatocellular damage leads to the elevation of liver enzymes. In a similar study conducted by Haleema Sadia et al., in 2019, which was done to investigate liver function test and renal function test in pregnant women affected with hypertensive disorder of pregnancy, it was found that ALT was elevated in 59(29.5%), total bilirubin was elevated in 60(30%) and 85(42.5%) thus it suggested that preeclampsia had deleterious effects on liver function test[74].

In our study, 59.1% (n=13) in group C and 10% (n=2) in group D had abnormal RFT. The p value is 0.001 which shows there is a significant relationship between RFT and hypertensive disorder of pregnancy. In a similar study conducted by Haleema Sadia et al., in 2019, which was done to investigate liver function test and renal function test in pregnant women

affected with hypertensive disorder of pregnancy, it was found that preeclampsia had deleterious effect on renal function test. There was increased urea in 46(23%), creatinine in 49(24.5%) and uric acid 91(45.5%) women[74]. In a similar study conducted by D Makuyana et al.,in 2002,on liver and kidney function tests in normal and preeclamptic geastation- a comparison with non-gestational reference values, it was found that renal indices, creatinine, urea, uric acid were significantly raised in preeclamptic women($p<0.001$)[75].

In our study, 63.6% (n=14) in group C and 30% (n=6) in group D, women had increased uric acid. The p value is 0.037 which shows a significant relationship between uric acid and hypertensive disease of pregnancy. In a similar study conducted by Naina Kumar et al., in 2019, which was done to study maternal serum uric acid as a predictor of severity of hypertensive disorders of pregnancy, it was observed that maternal serum uric acid levels ($p<0.05$) show a significant correlation with the severity of hypertensive disease of pregnancy[76]. The increase in serum uric acid levels in women with hypertensive disease of pregnancy is due to the impaired clearance from the kidneys, increase in tissue breakdown, acidosis and increased activity of xanthine oxidase/dehydrogenase enzyme[76,77,78].

In our study, 72.7%(n=16) in group C and 40% (n=8) in group D, women had increased lactate dehydrogenase (LDH).The p value is 0.032 which shows a significant relationship between LDH and hypertensive disease of pregnancy in our study. In a study conducted by Fernando Vazquez-Alaniz et al., in 2019, which was done to study lactate dehydrogenase in hypertensive disorders in pregnancy: severity or diagnosis marker; it was found that LDH is a significant factor indicative of cellular damage and endothelial dysfunction in HDP (p-0.037) . LDH can be used as a effective biochemical marker because it reflects the severity of the disease, occurrence of complications and fetal outcome[79].

In our study, 40.9% (n=9) in group C and 10% (n=2) in group D, women had mild hypertensive retinopathy. The p value is 0.023 which shows that there

is significant relationship between fundus and hypertensive disease of pregnancy in our study. Retinal vasospasm and resistance to blood flow is a possible explanation for visual symptoms. In a study conducted by Ibarra SAA et al., in 2016, which was done to study ophthalmological changes in pregnancy hypertensive disease, 78% women with HDP developed ophthalmological changes, it was concluded that retinopathy severity is associated with sustained hypertension in hypertensive disease of pregnancy. Fundoscopy permits to identify the evolution and severity of the illness so that the proper preventative measures could be taken[80].

In our study, 63.6% (n=14) in group C and 30% (n=6) in group D, women had preterm delivery. The p value is 0.029 which shows that there is statistically significant relationship between preterm delivery and hypertensive disease of pregnancy in our study. In a similar study by Juliette Madan et al., in 2010, which was done to study maternal obesity, gestational hypertension and preterm delivery, out of 58,112 pregnant women, 4653 women had preterm delivery. It was found that preterm delivery and cesarean section were more common in women who were having hypertensive disease of pregnancy[81]. It has been found that the obesity-prematurity relationship is complexly associated with hypertensive disorders of pregnancy that is playing crucial role[81].

In our study, 81.8% (n=18) in group C and 50% (n=10) in group D, women underwent cesarean section .The p value is 0.029 which shows that there is statistically significant relationship between cesarean section and hypertensive disease of pregnancy in our study. The increased prevalence of cesarean section could be due to accidental haemorrhages, placental infarction, intrauterine asphyxia, chronic placental insufficiency, fetal growth restriction, oligohydroamnios which leads to low Biophysical profile. In a similar study by Juliette Madan et al., in 2010, out of 5,375 deliveries, 66.3% were cesarean section, 33.7% were vaginal deliveries and forceps application was in 2.55%. It was found that preterm delivery and cesarean

section were more common in women who were having hypertensive disease of pregnancy[81].

In our study, 13.6% (n=3) in group C and 25% (n=5) in group D, women had application of forceps. The p value is 0.348 which shows that there is no statistically significant relationship between assisted delivery and hypertensive disease of pregnancy in our study. Forceps application can lead to lacerations, abrasions, ecchymosis, caput succedaneum, cephalhematoma, subarachnoid hemorrhage, brachial plexus lesion, facial paralysis, skull fracture, corneal compression injury, retinal hemorrhage, subgaleal hematoma so forceps application was less in our study in order to avoid these injuries[82].

In our study, 68.2% (n=15) in group C and 75% (n=15) in group D, women took antihypertensives thrice daily. The p value is 0.625 which shows that there is no statistically significant relationship between frequency of antihypertensive drug intake and hypertensive disease of pregnancy in our study. To the best of my knowledge no studies are available showing relationship between location of placenta and frequency of antihypertensive drug intake.

In our study, 77.3% (n=17) in group C and 75%(n=15) in group D, women took anti-hypertensives for 3 months. The p value is 0.228 which shows that there is no statistically significant relationship between duration of antihypertensive drug intake and hypertensive disease of pregnancy in our study. To the best of my knowledge no studies are available showing relationship between location of placenta and duration of antihypertensive drug intake.

NEONATAL OUTCOME

Women with lateral placenta who developed hypertension (group C) had higher rates of preterm deliveries at <37 weeks of gestation as compared to women with central placenta who developed hypertension (group D). In our study, 40% (n=16) in group C and 23.3% (n=14) in group D, neonates were preterm. The p-value was found to be 0.256 which was not significant.

Preterm births were more in group C probably due to higher rate of development of hypertension and associated complications. In a study conducted by Ibrahim A Abdelazim et al., in 2020, who studied the outcome of hypertensive disorders with pregnancy and found that there was significantly high relative risk and Odds ratio for preterm delivery PTD and low birth weight LBW in preeclampsia/superimposed preeclampsia group. (RR 2.1, OR;3.3;P=0.0001 and P=0.0001 respectively) (RR 2.01;OR;3.17;P=0.0001 and P=0.0001 respectively)[83].

Hypertensive disease of pregnancy is one of the leading cause of maternal mortality in developed and developing countries. It is also an important cause of perinatal morbidity and mortality and is strongly associated with fetal growth restriction leading to low birth weight. The process gets completely reversed after the delivery of the fetus and placenta but intrauterine growth retardation and prematurity are dangerous to the fetus and require delivery in a tertiary care centre. In our study, 68.2% (n=15) in group C and 35% (n=7) in group D, women were having low birth weight babies. In our study in group C more women had low birth weight as compared to group D. The p-value is 0.031 which showed it is statistically significant. Increased prevalence of low birth weight could be due to accidental haemorrhages, placental infarction, intrauterine asphyxia, chronic placental insufficiency. In a similar study conducted by Matthew Bridwell et al., in 2019, which was to study hypertensive disorders in pregnancy and maternal and neonatal outcome, it was found that in women with hypertensive disease of pregnancy, the adjusted odds ratio of having a low birth weight baby was four times higher that of women without hypertensive disease of pregnancy (aOR 4.17, 95% CI 3.19-5.45)[84].

In our study, 68.2% (n=15) in group C and 35% (n=7) in group D, neonates were female. The p-value is 0.031 which shows that there is significant association between sex and laterality of placenta for development of hypertension. In contrast, a study by Daniela Grigore et al., in 2008, which was done to study sex differences in the fetal programming of hypertension,

it was found that sex hormones might modulate the activity of regulatory systems leading to a lower incidence of hypertension and vascular dysfunction in females compared with males[85].

Obstetrical characteristics such as earlier gestational age at delivery, early gestational age at diagnosis of preeclampsia, lower neonatal birth weight lead to low APGAR score. In our study, the mean APGAR score at 1 minute in group C was 5.73 and in group D was 7.20. The p-value is 0.004 which is statistically significant. Low APGAR score is probably due to prematurity, hypoxia, drug effects. There was 18.2% (n=4) neonatal mortality in group C and no neonatal mortality in group D. In a similar study by Sulaeman A. Susilo et al., in 2014, it was found that early onset preeclampsia (adjusted OR=4.577;95% CI=2.147-9.757), haemolysis elevated liver enzymes, low platelet (HELLP) syndrome (adjusted OR=2.00;95%CI=1.38-2.91) were independent risk factors for having infant with low APGAR score at 1minute[86].

In our study, 86.4%(n=19) in group C and 50%(n=10) in group D, neonates got admitted in neonatal intensive care unit (NICU). The admission rate was higher in our study in group C owing to the fact that babies born to hypertensive mothers were premature, low birth weight and had complications like oligohydroamnios, FGR and abnormal Doppler. In a similar study conducted by Josje Langenveld et al., in 2011, it was found that babies from mothers with preeclampsia had an increased risk for admission to the neonatal intensive care unit when compared with children from normotensive mothers (odds ratio [OR],2.0;95% confidence interval [CI],(1.8-2.2)[87].

# SUMMARY AND CONCLUSION

This study was performed in the Department of Obstetrics and Gynecology, Dayanand Medical College and Hospital, Ludhiana to determine the location of placenta as a predictor of hypertensive disease of pregnancy.

In our study, in group C 45.5% (n=10) and in group D 50% (n=10) of women who developed hypertension belonged to age between 26-30 year. There was no statistically significant difference between the two groups. This implies that development of hypertension with lateral placenta as well as central placenta can occur in any age group.

The women who developed hypertension with lateral location of placenta as well as central placenta were overweight. However no significant relationship was found between BMI, location of placenta and hypertension.

The women who developed hypertension with lateral placenta as well as with central placenta were in urban residential areas. There was statistically significant relationship between laterality of placenta, development of hypertension and urban residential background.

The women who developed hypertension with lateral as well as central placenta were more in postgraduate group .However no statistically significant correlation was found between literacy, laterality of placenta and development of hypertension.

The women who developed hypertension with lateral placenta as well as with central placenta were mostly homemaker in our study. This was statistically significant which shows there is association between employment status, laterality of placenta and development of hypertension.

The development of hypertension in women with lateral placenta as well as with central placenta was more in primigravida. This was statistically significant which shows significant association between parity, laterality of placenta and development of hypertension.

The women who developed hypertension with both lateral placenta as well as with central placenta were mostly booked. However this was not statistically significant.

The women who developed hypertension with both lateral placenta as well as with central placenta had significant past history and family history of hypertension in previous pregnancy. This was statistically significant which showed association between development of hypertension, laterality of placenta and past as well as family history of hypertension in previous pregnancy.

The women who developed hypertension with both lateral placenta as well as central placenta mostly belonged to upper middle class and this was statistically significant. Thus significant association was found between development of hypertension, laterality of placenta and socioeconomic status.

The women with lateral placenta who developed hypertension had higher rate of maternal complications such as vomiting, headache, blurring of vision, pedal edema, generalized edema which were all found to be statistically significant. However in our study epigastric pain was not found to be a significant predictor of severity of hypertensive disease of pregnancy.

The women with lateral placenta who developed hypertension had higher rate of increased spot Protein: Creatinine ratio as compared to women with central placenta. Thus statistically significant association was found between development of hypertension, laterality of placenta and spot Protein: Creatinine ratio.

The women who developed hypertension with lateral placenta had higher rate of abnormal Renal function test and Liver function test, increased uric acid levels, increased Lactate dehydrogenase levels. This was statistically significant which shows association between abnormal renal function test, liver function test, increased uric acid level, increased lactate dehydrogenase level, laterality of placenta and development of hypertension.

The women who developed hypertension with lateral placenta had higher rate of mild hypertensive retinopathy on fundus examination than women with central placenta and it was statistically significant.

The women who developed hypertension with lateral placenta had statistically significant and higher rate of early preterm delivery than women with central placenta.

The women who developed hypertension with lateral placenta had statistically significant and higher rate of Cesarean section than women with central placenta.

The women who developed hypertension with central placenta had higher rates of application of forceps than women with lateral placenta but it was not statistically significant.

The women who developed hypertension with lateral placenta and central placenta took antihypertensive (labetalol) thrice daily (frequency) and took antihypertensive (labetalol) for 3 months (duration). However this was not statistically significant.

Women with lateral placenta who developed hypertension had higher rates of preterm deliveries (<37 weeks of gestation) as compared to women with central placenta.

In our study, neonates of women with lateral placenta had lower birth weight as compared to neonates of women with central placenta who developed hypertension and this was statistically significant.

In our study majority of neonate in group C were female while in group D were male. There was no significant association between sex of neonate and development of hypertension.

In our study, the mean APGAR score at 1 minute in group C was 5.73 and in group D was 7.20. The mean APGAR score at 1 minute in group C was lower than that of the group D which was statistically significant.

The NICU admission rate was higher in our study in group C as compared to group D owing to the fact that in group C more neonates were premature and had low birth weight.

In our study, in group A (with lateral placenta) 55% developed hypertension and in group B(with central placenta) 33.3% women developed hypertension. The p-value is 0.032 which came out to be statistically significant. Thus in our study significant relationship was found between lateral location of placenta and development of hypertension.

Based on all the observations, analysis and inferences it has been observed that the prevalence of hypertensive disease of pregnancy as well as it's complications(maternal and neonatal) are more in women with lateral location of placenta as compared to central placenta. So it is reasonable to consider that the location of placenta is a reliable predictor for development of hypertensive disease of pregnancy.

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| Sr. No | MRD NUMBER | Initials | AGE | Weight | Height | BMI | Area | DATE OF ADMISSION | ADMISSION NUMBER | EDUCATION | OCCUPATION | PARITY | PARITY | BOOKED/UNBOOKED | POG | LOCATION OF PLACENTA ON ULTRASOUND |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 612695 | P | 24 | 55.6 | 5'4'' | 21 | URBAN | 4/1/2019 | 2019081800 | GRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 18WEEKS | ANTERIOR |
| 2 | 364555 | AK | 28 | 67 | 5'4'' | 22.7 | RURAL | 10/1/2019 | 2019023898 | GRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2A1 | YES | 22WEEKS | ANTERIOR |
| 3 | 364555 | HK | 28 | 56 | 5'2'' | 22.6 | URBAN | 15/1/2019 | 2019023898 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 20WEEKS | ANTERIOR |
| 4 | 716078 | GR | 25 | 60 | 5'3'' | 23.4 | URBAN | 24/1/2019 | 2019027472 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 18WEEKS | LATERAL |
| 5 | 847414 | K | 38 | 60 | 5'5'' | 22 | RURAL | 28/1/2019 | 2019081144 | UNDERGRADUATE | HOSEWIFE | MULTIGRAVIDA | G4P3L2 | YES | 20+6WEEK | ANTERIOR |
| 6 | 713183 | R | 33 | 63 | 5'4'' | 23.8 | URBAN | 30/1/2019 | 2019081983 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4P1L1A2 | YES | 20WEEKS | ANTERIOR |
| 7 | 802941 | M | 30 | 76 | 5'4'' | 28.8 | URBAN | 2/2/2019 | 2019081345 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 24WEEKS | ANTERIOR |
| 8 | 848712 | RK | 34 | 80 | 5'2'' | 32.3 | RURAL | 5/2/2019 | 2019081040 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 19WEEKS | ANTERIOR |
| 9 | 845714 | S | 28 | 62 | 5'2'' | 26.7 | URBAN | 8/2/2019 | 2019080877 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 10 | 820040 | S | 29 | 90 | 5' | 34.4 | URBAN | 12/2/2019 | 2019080907 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 22WEEKS | ANTERIOR |
| 11 | 837627 | HK | 22 | 76 | 5'1'' | 25 | RURAL | 17/2/2019 | 2019075916 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3A2 | YES | 18WEEKS | POSTERIOR |
| 12 | 840308 | PK | 28 | 70 | 5'5'' | 22 | RURAL | 23/2/2019 | 2019075918 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 18WEEKS | ANTERIOR |
| 13 | 837803 | RK | 26 | 69 | 5'3'' | 25 | RURAL | 27/2/2019 | 2019076031 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 19WEEKS | POSTERIOR |
| 14 | 686287 | SK | 24 | 59 | 5'1'' | 24.6 | RURAL | 1/3/2019 | 2019076036 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | POSTERIOR |
| 15 | 837533 | S | 32 | 102 | 5'6'' | 32 | RURAL | 3/3/2019 | 2019076038 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 18WEEKS | POSTERIOR |
| 16 | 602785 | JK | 29 | 65 | 4'8'' | 24.7 | RURAL | 6/3/2019 | 2019026449 | POSTGRADUATE | TEACHER | MULTIGRAVIDA | G2P1L0 | YES | 18+4 | POSTERIOR |
| 17 | 716186 | MG | 31 | 79 | 5'4'' | 22.7 | URBAN | 10/3/2019 | 2019027545 | GRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 20WEEKS | POSTERIOR |
| 18 | 714540 | KK | 29 | 60 | 5' | 25 | URBAN | 15/3/2019 | 2019026923 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 22+2WEEK | ANTERIOR |
| 19 | 120801 | KB | 39 | 72 | 5'2'' | 26.5 | URBAN | 17/3/2019 | 2019027730 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | ANTERIOR |
| 20 | 640008 | A | 26 | 50 | 5'4'' | 18.9 | URBAN | 19/3/2019 | 2019027229 | GRADUATE | TEACHER | PRIMIGRAVIDA | G1 | YES | 19WEEKS | ANTERIOR |
| 21 | 713018 | RL | 31 | 77 | 5'1'' | 28.2 | RURAL | 23/3/2019 | 2019026285 | POSTGRADUATE | TEACHER | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 22 | 7921 | DK | 22 | 77.8 | 5'2'' | 31.4 | URBAN | 25/3/2019 | 2019023388 | GRADUATE | STAFF NURSE | PRIMIGRAVIDA | G1 | YES | 20+4WEEK | LATERAL |
| 23 | 705147 | AK | 22 | 77 | 5'3'' | 27.3 | RURAL | 28/3/2019 | 2019023396 | GRADUATE | HOSEWIFE | PRIMIGRAVIDA | G1 | YES | 20+2WEEKS | ANTERIOR |
| 24 | 565864 | N | 28 | 60 | 5'2'' | 24.2 | URBAN | 5/4/2019 | 2019024065 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | ANTERIOR |
| 25 | 708573 | A | 30 | 100.1 | 5'6'' | 32 | URBAN | 9/4/2019 | 2019024626 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 22+3WEEK | POSTERIOR |
| 26 | 704144 | MR | 28 | 70 | 5'5'' | 25.7 | URBAN | 12/4/2019 | 2019022907 | POSTGRADUATE | TEACHER | MULTIGRAVIDA | G3P1L1A1 | YES | 24WEEKS | ANTERIOR |
| 27 | 571394 | PK | 29 | 56 | 5'7'' | 19.3 | URBAN | 16/4/2019 | 2019024608 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 18WEEKS | POSTERIOR |
| 28 | 661119 | SK | 48 | 64 | 5'1'' | 25 | URBAN | 19/4/2019 | 2019006482 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4P1A2 | YES | 22+1WEEK | POSTERIOR |
| 29 | 541325 | SM | 28 | 71 | 5'2'' | 28.6 | URBAN | 22/4/2019 | 2019023110 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2A1 | YES | 23WEEK | POSTERIOR |
| 30 | 800260 | RK | 26 | 70 | 5'7'' | 24.2 | URBAN | 26/4/2019 | 2019060262 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 31 | 801458 | RK | 26 | 62 | 5' | 26.7 | RURAL | 29/4/2019 | 2019060834 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |
| 32 | 792594 | J | 36 | 62 | 5'2'' | 26.9 | URBAN | 1/5/2019 | 2019057442 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3P2L2 | YES | 24WEEKS | LATERAL |
| 33 | 750480 | AK | 33 | 104 | 5'8'' | 31.5 | RURAL | 5/5/2019 | 2019040990 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 22WEEKS | POSTERIOR |
| 34 | 835322 | G | 31 | 60 | 5'5'' | 22 | RURAL | 8/5/2019 | 2019074817 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3P1L1A1 | YES | 22WEEKS | ANTERIOR |
| 35 | 836097 | HK | 20 | 59 | 5'3'' | 23 | URBAN | 15/5/2019 | 2019075095 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | ANTERIOR |
| 36 | 832838 | P | 30 | 90 | 5'5'' | 33 | RURAL | 26/5/2019 | 2019075088 | POSTGRADUATE | STAFF NURSE | MULTIGRAVIDA | G5A4 | YES | 22WEEKS | ANTERIOR |
| 37 | 484927 | SK | 27 | 64 | 5'1'' | 26.7 | URBAN | 28/5/2019 | 2019074497 | GRADUATE | TEACHER | MULTIGRAVIDA | G3A2 | YES | 23WEEK | POSTERIOR |
| 38 | 858044 | RK | 34 | 83 | 5'3'' | 27.3 | URBAN | 30/5/2019 | 2019085305 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 18WEEKS | ANTERIOR |
| 39 | 450551 | S | 29 | 69 | 5'2'' | 24.2 | URBAN | 1/6/2019 | 2019085611 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 40 | 859391 | L | 24 | 49 | 5'3'' | 19.1 | URBAN | 10/6/2019 | 2019085918 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |
| 41 | 618016 | S | 27 | 62 | 5' | 26.7 | URBAN | 18/6/2019 | 2019086207 | GRADUATE | LAB.TECHNICIAN | PRIMIGRAVIDA | G1 | YES | 18WEEKS | POSTERIOR |
| 42 | 711775 | RD | 30 | 112 | 5'7'' | 31.1 | URBAN | 26/6/2019 | 2019085401 | GRADUATE | CLERK | MULTIGRAVIDA | G2P1L1 | YES | 20+4WEEKS | ANTERIOR |
| 43 | 857581 | SD | 24 | 85 | 5' | 26.5 | URBAN | 6/7/2019 | 2019085111 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2A1 | YES | 19WEEKS | POSTERIOR |
| 44 | 850065 | M | 22 | 94 | 5'8'' | 26.8 | URBAN | 18/7/2019 | 2019081609 | POSTGRADUATE | TEACHER | PRIMIGRAVIDA | G1 | YES | 18+6WEEKS | LATERAL |
| 45 | 775214 | S | 25 | 50 | 5'2'' | 20.2 | URBAN | 27/7/2019 | 2019089425 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 18+8WEEKS | ANTERIOR |
| 46 | 866673 | B | 32 | 74 | 5'1'' | 30.8 | RURAL | 4/8/2019 | 2019089206 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20+2WEEKS | POSTERIOR |
| 47 | 172189 | SA | 28 | 65 | 5'2'' | 24.2 | URBAN | 14/8/2019 | 2019089192 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4P1L1A2 | YES | 18+6WEEKS | POSTERIOR |
| 48 | 770592 | HK | 45 | 68 | 5'3'' | 23.4 | URBAN | 19/8/2019 | 2019088961 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 18WEEKS | ANTERIOR |
| 49 | 718218 | PK | 31 | 65 | 5'3'' | 24.6 | URBAN | 20/8/2019 | 2019088703 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2A1 | YES | 20WEEKS | POSTERIOR |
| 50 | 796664 | NK | 22 | 112 | 5'4'' | 37.8 | URBAN | 22/8/2019 | 2019088928 | GRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 19WEEKS | POSTERIOR |
| 51 | 729147 | A | 25 | 85 | 5'7'' | 26.9 | URBAN | 25/8/2019 | 2019089330 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2A1 | YES | 18WEEKS | POSTERIOR |
| 52 | 882761 | S | 30 | 80 | 5'2'' | 26.5 | URBAN | 28/8/2019 | 2020003931 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4P3L1 | UNBOOKED | 21WEEK | ANTERIOR |
| 53 | 869544 | H | 33 | 66 | 5'3'' | 22.7 | RURAL | 2/9/2019 | 2020004181 | POSTGRADUATE | TEACHER | MULTIGRAVIDA | G2P1L1 | UNBOOKED | 20WEEKS | POSTERIOR |
| 54 | 881584 | CK | 34 | 65 | 5'4'' | 24.6 | RURAL | 5/9/2019 | 2020003382 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3P2L2 | UNBOOKED | 22WEEKS | ANTERIOR |
| 55 | 882183 | AC | 32 | 55 | 5'2'' | 20.2 | URBAN | 8/9/2019 | 2020003666 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 18WEEKS | POSTERIOR |
| 56 | 835627 | AG | 33 | 67 | 5' | 24.2 | URBAN | 10/9/2019 | 2020003703 | GRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 20WEEKS | ANTERIOR |
| 57 | 882216 | SK | 32 | 65 | 5'3'' | 23.4 | RURAL | 13/9/2019 | 2020003696 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 21WEEK | ANTERIOR |
| 58 | 882798 | HK | 24 | 70 | 5'3'' | 21.5 | RURAL | 16/9/2019 | 2020003953 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4A3 | YES | 19WEEKS | POSTERIOR |
| 59 | 881683 | M | 39 | 88 | 5'2'' | 24.6 | URBAN | 18/9/2019 | 2020003473 | GRADUATE | TEACHER | MULTIGRAVIDA | G2P1L1 | YES | 20WEEKS | POSTERIOR |
| 60 | 882198 | A | 29 | 65 | 5'4'' | 21.9 | URBAN | 21/9/2019 | 2020003685 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2A1 | YES | 18WEEKS | ANTERIOR |
| 61 | 889365 | A | 35 | 76 | 5'1'' | 24.2 | RURAL | 24/9/2019 | 2020006871 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4P2L1A1 | YES | 22WEEKS | POSTERIOR |
| 62 | 889398 | MR | 35 | 70 | 5'4'' | 22.7 | URBAN | 27/9/2019 | 2020006897 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2A1 | YES | 20WEEKS | LATERAL |
| 63 | 824046 | AK | 35 | 70 | 5'2' | 24.2 | RURAL | 28/9/2019 | 2020005836 | POSTGRADUATE | TEACHER | PRIMIGRAVIDA | G1 | YES | 22WEEKS | ANTERIOR |
| 64 | 874245 | NP | 20 | 48 | 4'10'' | 25.9 | URBAN | 2/10/2019 | 2020006470 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | NO | 21WEEKS | LATERAL |
| 65 | 885338 | PK | 35 | 58 | 5'3'' | 19.5 | URBAN | 5/10/2019 | 2020005159 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | POSTERIOR |
| 66 | 885502 | KJ | 30 | 82 | 5'5'' | 30.3 | URBAN | 7/10/2019 | 2020005232 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |

| Sr. No | MRD NUMBER | CALCIFICATION OF PLACENTA ON ULTRASOUND | ULTRASOUND FOR LOCATION OF PLACENTA | PAST HISTORY | FAMILY HISTORY | SOCIO-ECONOMIC HISTORY | SOCIOECONOMIC STATUS | NUTRITIONAL HISTORY | PERIOD OF GESTATION AT THE TIME OF DELIVERY |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 612695 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 36WEEKS |
| 2 | 364555 | NO | 22WEEKS | NO RELEVANT | HTN | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 38+5WEEKS |
| 3 | 364555 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 38+3WEEKS |
| 4 | 716078 | NO | 18WEEKS | HTN | HTN | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 36WEEKS |
| 5 | 847414 | NO | 20+6WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 34+3WEEKS |
| 6 | 713183 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 39WEEKS |
| 7 | 802941 | NO | 21WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 33+6WEEK |
| 8 | 848712 | NO | 19WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 35+1WEEK |
| 9 | 845714 | NO | 20WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 36+2WEEK |
| 10 | 820040 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 40+4 WEEK |
| 11 | 837627 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 37WEEKS |
| 12 | 840308 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 30+4WEEKS |
| 13 | 837803 | NO | 19WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 33+4WEEKS |
| 14 | 686287 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 39+6WEEKS |
| 15 | 837533 | NO | 18WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 29+2WEEKS |
| 16 | 602785 | NO | 18+4WEEK | HTN | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 33+6WEEKS |
| 17 | 716186 | YES | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 36+1WEEK |
| 18 | 714540 | NO | 22+2WEEK | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 34WEEKS |
| 19 | 120801 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 34WEEKS |
| 20 | 640008 | NO | 19WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 36+2WEEK |
| 21 | 713018 | NO | 20WEEKS | HTN | HTN | ADEQUATE | UPPER CLASS | ADEQUATE | 36+2WEEK |
| 22 | 7921 | NO | 20+4WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 38WEEKS |
| 23 | 705147 | NO | 20+2WEEK | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 32+3WEEKS |
| 24 | 565864 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 40WEEKS |
| 25 | 708573 | NO | 22+3WEEKS | HYPOTHYROID | HTN | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 30WEEKS |
| 26 | 704144 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 30+2WEEKS |
| 27 | 571394 | NO | 18WEEKS | NO RELEVANT | HTN | ADEQUATE | UPPER CLASS | ADEQUATE | 34WEEKS |
| 28 | 661119 | NO | 20+2WEEK | DM | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 34+4WEEKS |
| 29 | 541325 | NO | 23WEEKS | NO RELEVANT | HTN,DM | ADEQUATE | UPPER CLASS | ADEQUATE | 36WEEKS |
| 30 | 800260 | NO | 20WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 35+2WEEKS |
| 31 | 801458 | NO | 22WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 39+3WEEKS |
| 32 | 792594 | NO | 23WEEKS | HTN | HTN | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 30WEEKS |
| 33 | 750480 | NO | 21WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 35+5WEEKS |
| 34 | 835322 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 40+2WEEKS |
| 35 | 836097 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 34+5WEEKS |
| 36 | 832838 | NO | 22WEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 38WEEKS |
| 37 | 484927 | NO | 23WEEKS | HTN | HTN | ADEQUATE | LOWER CLASS | ADEQUATE | 37WEEKS |
| 38 | 858044 | NO | 18 WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 37WEEKS |
| 39 | 450551 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 39+4WEEKS |
| 40 | 859391 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 39WEEKS |
| 41 | 618016 | NO | 18WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 38+5WEEKS |
| 42 | 711775 | NO | 20+4WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 40+2WEEKS |
| 43 | 857581 | NO | 19WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 32WEEKS |
| 44 | 850065 | NO | 18+6WEEKS | HTN | HTN | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 34WEEKS |
| 45 | 775214 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 40WEEKS |
| 46 | 866673 | NO | 20+2WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 40+1WEEKS |
| 47 | 172189 | NO | 18+6WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 36+1WEEK |
| 48 | 770592 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 38+3WEEKS |
| 49 | 718218 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 40WEEKS |
| 50 | 796664 | NO | 19WEEKS | NO RELEVANT | DM,HYPOTHYROIDISM | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 39+6WEEKS |
| 51 | 729147 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 39+6WEEKS |
| 52 | 882761 | NO | 21WEEKS | NO RELEVANT | HTN,DM,HYPOTHYROID | ADEQUATE | LOWER CLASS | ADEQUATE | 34+3WEEKS |
| 53 | 869544 | NO | 20WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 37+5WEEKS |
| 54 | 881584 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 36+3WEEKS |
| 55 | 882183 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 36+5WEEKS |
| 56 | 835627 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 37+5WEEKS |
| 57 | 882216 | NO | 21WEEKS | HTN,DM,THYROID | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 38+3WEEKS |
| 58 | 882798 | NO | 19WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 32+1WEEKS |
| 59 | 881683 | NO | 20WEEKS | HTN,DM,HYPOTHROID | HTN,DM | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 33+4WEEKS |
| 60 | 882198 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 39+1WEEK |
| 61 | 889365 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 35+6WEEKS |
| 62 | 889398 | NO | 20WEEKS | HTN | HTN | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 36WEEKS |
| 63 | 824046 | NO | 22WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 37+2WEEK |
| 64 | 874245 | NO | 21WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 40+3WEEKS |
| 65 | 885338 | NO | 20WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 32+6WEEKS |
| 66 | 885502 | NO | 22WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 34+6WEEKS |

| Sr. No | MRD NUMBER | PRETERM / TERM | MODE OF DELIVERY | VAGINAL (WITH FORCEPS/WITHOUT FORCEPS CAESAREAN | MATERNAL COMPLICATIONS_VOMITING | HEADACHE | BLURRING OF VISION | EPIGASTRIC PAIN | PULSE | SBP | DBP |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 612695 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 92 | 140 | 100 |
| 2 | 364555 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 96 | 130 | 90 |
| 3 | 364555 | TERM | NVD | NVD WITH RMLE | YES | NO | NO | NO | 90 | 140 | 90 |
| 4 | 716078 | LATE PRETERM | NVD | NVD WITH RMLE | YES | YES | NO | NO | 88 | 150 | 90 |
| 5 | 847414 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 140 | 90 |
| 6 | 713183 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 78 | 150 | 100 |
| 7 | 802941 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 100 |
| 8 | 848712 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 78 | 140 | 90 |
| 9 | 845714 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | YES | YES | 84 | 150 | 100 |
| 10 | 820040 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 86 | 150 | 90 |
| 11 | 837627 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 82 | 140 | 90 |
| 12 | 840308 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 150 | 90 |
| 13 | 837803 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 90 |
| 14 | 686287 | TERM | NVD | NVD WITH RMLE | NO | NO | NO | NO | 84 | 140 | 90 |
| 15 | 837533 | EARLY PRETERM | NVD | NVD WITH RMLE | YES | NO | YES | YES | 89 | 160 | 100 |
| 16 | 602785 | EARLY PRETERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 98 | 140 | 90 |
| 17 | 716186 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 86 | 150 | 100 |
| 18 | 714540 | EARLY PRETERM | NVD | NVD WITH RMLE WITH FORCEPS | NO | YES | NO | NO | 84 | 190 | 110 |
| 19 | 120801 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 90 |
| 20 | 640008 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 90 | 150 | 90 |
| 21 | 713018 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | YES | NO | 94 | 140 | 90 |
| 22 | 7921 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 90 | 150 | 90 |
| 23 | 705147 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 92 | 140 | 90 |
| 24 | 565864 | TERM | NVD | NVD WITH RMLE | NO | NO | NO | NO | 96 | 150 | 90 |
| 25 | 708573 | EARLY PRETERM | NVD | NVD WITH RMLE | YES | YES | YES | YES | 98 | 150 | 100 |
| 26 | 704144 | EARLY PRETERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 80 | 150 | 90 |
| 27 | 571394 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 90 | 140 | 90 |
| 28 | 661119 | LATE PRETERM | NVD | NVD WITH RMLE | YES | NO | NO | NO | 82 | 140 | 90 |
| 29 | 541325 | LATE PRETERM | NVD | NVD WITH RMLE | YES | NO | NO | NO | 86 | 150 | 100 |
| 30 | 800260 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 76 | 140 | 90 |
| 31 | 801458 | TERM | NVD | NVD WITH RMLE WITH FORCEPS | YES | YES | NO | NO | 96 | 150 | 100 |
| 32 | 792594 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | YES | YES | 80 | 140 | 90 |
| 33 | 750480 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 90 | 160 | 90 |
| 34 | 835322 | TERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 84 | 150 | 100 |
| 35 | 836097 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 86 | 140 | 90 |
| 36 | 832838 | TERM | NVD | NVD WITH RMLE WITH FORCEPS | YES | NO | NO | NO | 92 | 150 | 90 |
| 37 | 484927 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 82 | 140 | 90 |
| 38 | 858044 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 90 |
| 39 | 450551 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 94 | 150 | 90 |
| 40 | 859391 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 72 | 150 | 90 |
| 41 | 618016 | TERM | NVD | NVD WITH RMLE | YES | NO | NO | NO | 86 | 140 | 90 |
| 42 | 711775 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 82 | 140 | 90 |
| 43 | 857581 | EARLY PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 84 | 150 | 90 |
| 44 | 850065 | EARLY PRETERM | NVD | NVD WITH RMLE | YES | NO | YES | NO | 88 | 140 | 90 |
| 45 | 775214 | TERM | NVD | NVD WITH RMLE WITH FORCEPS | NO | NO | NO | NO | 90 | 140 | 90 |
| 46 | 866673 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 84 | 150 | 90 |
| 47 | 172189 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 90 |
| 48 | 770592 | TERM | LSCS | CAESSAREAN | NO | YES | NO | NO | 74 | 150 | 90 |
| 49 | 718218 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 140 | 90 |
| 50 | 796664 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 90 |
| 51 | 729147 | TERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 84 | 150 | 90 |
| 52 | 882761 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 82 | 140 | 90 |
| 53 | 869544 | TERM | NVD | NVD WITH RMLE | YES | NO | NO | NO | 84 | 140 | 100 |
| 54 | 881584 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 98 | 140 | 90 |
| 55 | 882183 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 140 | 90 |
| 56 | 835627 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 140 | 90 |
| 57 | 882216 | TERM | NVD | NVD WITH RMLE | YES | NO | NO | NO | 88 | 150 | 90 |
| 58 | 882798 | EARLY PRETERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 82 | 140 | 90 |
| 59 | 881683 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 94 | 140 | 90 |
| 60 | 882198 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 98 | 150 | 90 |
| 61 | 889365 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 88 | 140 | 90 |
| 62 | 889398 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | YES | 84 | 140 | 90 |
| 63 | 824046 | TERM | LSCS | CAESSAREAN | NO | YES | NO | NO | 88 | 160 | 100 |
| 64 | 874245 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 150 | 90 |
| 65 | 885338 | EARLY PRETERM | NVD | NVD WITH RMLE | YES | YES | NO | NO | 82 | 140 | 90 |
| 66 | 885502 | LATE PRETERM | NVD | NVD WITH RMLE WITH FORCEPS | YES | YES | YES | NO | 86 | 140 | 90 |

| Sr. No | MRD NUMBER | RESPIRATORY RATE | TEMPERATURE | EDEMA_GENERALISED | EDEMA_PEDAL | SBP_18HRS | SBP_20 HRS | SBP_22 HRS | SBP_24 HRS | SBP_28 HRS | SBP_32 HRS | SBP_36 HRS | SBP_40 HRS | PULSE_18HRS | PULSE_20 | PULSE_22 | PULSE_24 | PULSE_28 | PULSE_32 | PULSE_36 | PULSE | RR_18HRS | RR_20 | RR_22 | RR_24 | RR_28 | RR_32 | RR_36 | RR_40 | HTN |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 612695 | 22 | 98.7 | NO | YES | 120/80 | 130/80 | 130/90 | 140/90 | 150/90 | 140/90 | 140/100 | | 84 | 86 | 90 | 92 | 94 | 96 | 98 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 2 | 364555 | 20 | 98 | NO | NO | 120/80 | 130/80 | 130/80 | 120/80 | 130/80 | 140/90 | 130/80 | | 80 | 84 | 86 | 82 | 80 | 86 | 88 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 3 | 364555 | 22 | 98.6 | NO | NO | 120/80 | 130/80 | 130/90 | 130/90 | 140/80 | 140/90 | 140/90 | | 72 | 74 | 76 | 80 | 72 | 74 | 76 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 4 | 716078 | 21 | 98.7 | YES | NO | 140/90 | 120/80 | 130/80 | 130/80 | 140/90 | 140/90 | 130/81 | | 88 | 92 | 94 | 96 | 98 | 99 | 94 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | YES |
| 5 | 847414 | 22 | 98.8 | N | NO | 110/70 | 120/80 | 130/90 | 130/90 | 140/90 | 130/90 | | | 82 | 80 | 86 | 88 | 80 | 72 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 6 | 713183 | 22 | 98.6 | NO | NO | 120/80 | 130/90 | 120/80 | 130/80 | 140/90 | 130/190 | 130/90 | | 82 | 80 | 74 | 72 | 86 | 92 | 94 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | NO |
| 7 | 802941 | 24 | 98.7 | NO | NO | 110/70 | 120/90 | 130/90 | 120/90 | 140/90 | 140/90 | | | 80 | 82 | 84 | 82 | 80 | 86 | | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | 24 | NO |
| 8 | 848712 | 22 | 98.6 | NO | NO | 110/70 | 120/80 | 130/80 | 120/80 | 140/90 | 150/90 | | | 72 | 74 | 76 | 78 | 80 | 84 | | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | NO |
| 9 | 845714 | 22 | 98.7 | YES | YES | 130/70 | 130/80 | 140/90 | 140/90 | 150/90 | 140/90 | 140/90 | | 82 | 86 | 84 | 82 | 78 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | YES |
| 10 | 820040 | 22 | 98.8 | NO | NO | 120/70 | 130/80 | 130/80 | 130/80 | 140/90 | 130/80 | 140/90 | 140/90 | 80 | 84 | 86 | 88 | 82 | 80 | 82 | 84 | 20 | 20 | 20 | 20 | 20 | 20 | 20 | 20 | NO |
| 11 | 837627 | 21 | 98.7 | NO | NO | 110/70 | 120/80 | 130/80 | 120/80 | 140/90 | 130/80 | 130/80 | 130/90 | 82 | 81 | 82 | 84 | 82 | 84 | 82 | | 21 | 21 | 21 | 21 | 21 | 21 | 21 | 21 | NO |
| 12 | 840308 | 22 | 98.6 | YES | YES | 110/70 | 120/80 | 130/90 | 130/80 | 140/90 | | | | 72 | 84 | 80 | 82 | 86 | | | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | NO |
| 13 | 837803 | 24 | 98.7 | NO | NO | 130/82 | 120/80 | 130/90 | 120/80 | 140/90 | 130/90 | | | 84 | 88 | 86 | 88 | 86 | 88 | | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | 24 | NO |
| 14 | 686287 | 22 | 98.8 | NO | NO | 120/80 | 130/80 | 130/80 | 130/80 | 140/90 | 140/100 | 130/80 | | 72 | 74 | 76 | 78 | 80 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | NO |
| 15 | 837533 | 22 | 98.6 | YES | NO | 130/80 | 120/80 | 130/80 | 130/80 | 140/90 | | | | 84 | 82 | 86 | 98 | 94 | | | | 22 | 22 | 22 | 22 | 22 | | | | YES |
| 16 | 602785 | 20 | 98.4 | NO | NO | 130/80 | 130/80 | 120/80 | 130/80 | 140/90 | 140/90 | | | 80 | 86 | 82 | 84 | 88 | 86 | | | 20 | 20 | 20 | 20 | 20 | 20 | | | YES |
| 17 | 716186 | 22 | 98.7 | NO | YES | 120/80 | 130/80 | 130/90 | 130/90 | 130/80 | 140/90 | 140/100 | | 82 | 84 | 86 | 82 | 80 | 84 | 82 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | NO |
| 18 | 714540 | 20 | 98/6 | YES | YES | 130/80 | 120/80 | 130/80 | 140/90 | 130/90 | 150/100 | | | 84 | 82 | 80 | 82 | 80 | 78 | | | 20 | 20 | 20 | 20 | 20 | 20 | | | YES |
| 19 | 120801 | 22 | 98.6 | NO | NO | 120/78 | 130/80 | 130/80 | 140/80 | 130/90 | 130/80 | 140/90 | | 74 | 78 | 72 | 86 | 84 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 20 | 640008 | 24 | 98.8 | NO | YES | 130/80 | 130/80 | 120/80 | 130/90 | 140/90 | 130/90 | 140/100 | | 74 | 78 | 76 | 70 | 80 | 82 | 84 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | YES |
| 21 | 713018 | 22 | 98.7 | YES | YES | 130/80 | 130/90 | 140/90 | 140/90 | 140/96 | 150/90 | 140/90 | | 72 | 74 | 70 | 72 | 74 | 76 | 78 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 22 | 7921 | 20 | 98.6 | YES | NO | 110/70 | 130/90 | 140/90 | 150/100 | 140/90 | 130/90 | 140/90 | | 88 | 84 | 82 | 80 | 88 | 82 | 86 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | YES |
| 23 | 705147 | 22 | 98.7 | YES | YES | 130/70 | 130/80 | 130/90 | 140/90 | 130/90 | 140/90 | | | 82 | 84 | 86 | 88 | 90 | 98 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 24 | 565864 | 22 | 97.8 | NO | NO | 120/80 | 130/90 | 140/80 | 130/80 | 140/90 | 130/80 | 140/80 | 130/90 | 88 | 86 | 84 | 88 | 90 | 92 | 94 | 98 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | NO |
| 25 | 708573 | 22 | 97.2 | YES | NO | 130/70 | 130/80 | 130/90 | 140/90 | 150/100 | | | | 80 | 78 | 94 | 92 | 98 | | | | 22 | 20 | 22 | 22 | 20 | | | | YES |
| 26 | 704144 | 22 | 98.3 | NO | NO | 130/80 | 120/80 | 140/80 | 130/90 | 140/90 | | | | 82 | 88 | 84 | 88 | 98 | | | | 22 | 22 | 22 | 22 | 22 | | | | YES |
| 27 | 571394 | 22 | 98.6 | NO | NO | 120/80 | 130/80 | 130/90 | 140/80 | 140/90 | 150/100 | | | 72 | 78 | 76 | 78 | 84 | 88 | | | 20 | 20 | 20 | 20 | 20 | | | | NO |
| 28 | 661119 | 22 | 98.7 | YES | NO | 130/80 | 130/80 | 130/80 | 140/80 | 140/90 | 140/80 | | | 82 | 86 | 90 | 84 | 88 | 82 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 29 | 541325 | 22 | 97 | NO | NO | 110/80 | 120/80 | 130/70 | 140/90 | 140/84 | 140/90 | 150/100 | | 80 | 88 | 90 | 82 | 86 | 84 | 92 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 30 | 800260 | 24 | 98.7 | YES | YES | 130/80 | 110/70 | 140/90 | 150/90 | 140/90 | 140/100 | | | 76 | 78 | 80 | 76 | 80 | 90 | | | 24 | 24 | 24 | 24 | 24 | 24 | | | YES |
| 31 | 801458 | 20 | 98 | NO | YES | 130/80 | 130/90 | 140/90 | 140/90 | 140/90 | 130/90 | 140/90 | | 98 | 94 | 96 | 98 | 94 | 96 | 98 | | 20 | 20 | 20 | 20 | 20 | 20 | | | YES |
| 32 | 792594 | 22 | 98.8 | YES | YES | 110/70 | 120/80 | 130/80 | 140/90 | 140/90 | | | | 70 | 72 | 74 | 78 | 80 | | | | 20 | 20 | 20 | 22 | 22 | | | | YES |
| 33 | 750480 | 20 | 97.8 | YES | NO | 130/80 | 130/80 | 140/80 | 140/90 | 140/88 | 160/90 | 200/100 | | 104 | 100 | 102 | 104 | 102 | 104 | 102 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | YES |
| 34 | 835322 | 22 | 98.7 | YES | YES | 120/80 | 130/80 | 130/90 | 140/80 | 140/90 | 140/90 | 130/80 | 140/90 | 74 | 76 | 72 | 78 | 72 | 76 | 78 | 74 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | NO |
| 35 | 836097 | 20 | 98.2 | YES | YES | 120/80 | 130/90 | 140/80 | 130/80 | 140/90 | 140/80 | | | 84 | 86 | 84 | 86 | 84 | 82 | | | 20 | 20 | 20 | 20 | 20 | 20 | | | NO |
| 36 | 832838 | 22 | 98.7 | YES | YES | 130/80 | 130/90 | 140/80 | 140/80 | 140/90 | 130/80 | 140/80 | | 96 | 98 | 92 | 90 | 84 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 37 | 484927 | 22 | 98.6 | YES | NO | 130/70 | 120/80 | 130/80 | 140/80 | 140/92 | 140/80 | 130/80 | | 72 | 78 | 80 | 84 | 86 | 88 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 38 | 858044 | 24 | 98.8 | YES | YES | 110/80 | 120/80 | 130/80 | 130/80 | 140/90 | 130/80 | 140/80 | | 80 | 82 | 84 | 86 | 80 | 82 | 84 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | NO |
| 39 | 450551 | 20 | 98.7 | NO | YES | 130/80 | 120/80 | 130/80 | 140/90 | 140/90 | 130/80 | 140/80 | | 82 | 84 | 76 | 78 | 80 | 82 | 84 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | YES |
| 40 | 859391 | 22 | 98,8 | NO | NO | 120/80 | 130/90 | 140/80 | 130/80 | 140/90 | 140/80 | 130/80 | | 70 | 74 | 78 | 80 | 82 | 84 | 72 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 41 | 618016 | 22 | 98.7 | NO | NO | 130/70 | 120/80 | 130/80 | 140/80 | 150/100 | 130/80 | 140/80 | | 80 | 82 | 84 | 88 | 80 | 82 | 86 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 42 | 711775 | 20 | 98.8 | YES | YES | 120/80 | 130/80 | 140/80 | 140/80 | 140/90 | 140/80 | 130/80 | 140/80 | 80 | 86 | 82 | 86 | 82 | 84 | 89 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 43 | 857581 | 22 | 98.7 | NO | YES | 130/70 | 110/70 | 140/80 | 130/80 | 140/90 | 140/80 | | | 84 | 90 | 98 | 96 | 98 | 88 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 44 | 850065 | 22 | 98.8 | NO | NO | 130/80 | 130/80 | 140/90 | 140/100 | 140/90 | 150/100 | | | 80 | 84 | 86 | 82 | 88 | 90 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | YES |
| 45 | 775214 | 24 | 98.6 | NO | YES | 130/80 | 130/70 | 140/80 | 140/80 | 150/100 | 140/90 | 140/80 | 140/80 | 84 | 88 | 86 | 84 | 80 | 82 | 88 | 82 | 24 | 24 | 24 | 24 | 24 | 24 | 24 | 24 | YES |
| 46 | 866673 | 22 | 98.8 | NO | NO | 130/80 | 130/80 | 140/80 | 130/80 | 140/100 | 140/80 | 130/80 | 140/80 | 84 | 80 | 86 | 88 | 82 | 84 | 88 | 90 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | YES |
| 47 | 172189 | 20 | 98.7 | NO | NO | 110/70 | 120/80 | 130/80 | 140/80 | 140/90 | 130/80 | 140/80 | | 84 | 82 | 86 | 80 | 82 | 88 | 82 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 48 | 770592 | 22 | 98.6 | NO | NO | 130/98 | 130/80 | 140/90 | 140/80 | 140/90 | 130/80 | 140/82 | | 82 | 80 | 84 | 80 | 82 | 88 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 49 | 718218 | 22 | 98.8 | NO | YES | 120/80 | 130/80 | 140/80 | 140/80 | 140/80 | 140/80 | 140/80 | 140/80 | 82 | 88 | 80 | 84 | 86 | 80 | 82 | 84 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 50 | 796664 | 20 | 98.6 | NO | NO | 120/70 | 130/70 | 140/80 | 130/80 | 140/80 | 130/80 | 130/70 | | 84 | 82 | 80 | 88 | 84 | 86 | 88 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 51 | 729147 | 22 | 98.8 | NO | NO | 130/90 | 110/70 | 130/80 | 140/80 | 130/70 | 140/80 | 140/80 | | 88 | 86 | 84 | 82 | 90 | 92 | 86 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 52 | 882761 | 22 | 98.4 | NO | NO | 120/80 | 130/80 | 140/80 | 140/80 | 140/90 | 150/100 | | | 84 | 88 | 86 | 82 | 84 | 82 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 53 | 869544 | 24 | 98.6 | NO | NO | 130/80 | 130/80 | 140/80 | 140/80 | 150/100 | 140/90 | 140/90 | | 76 | 78 | 86 | 82 | 84 | 80 | 84 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | YES |
| 54 | 881584 | 22 | 98.8 | NO | NO | 120/80 | 130/80 | 140/80 | 140/80 | 150/100 | 130/84 | 150/100 | | 84 | 86 | 88 | 82 | 84 | 88 | 86 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 55 | 882183 | 22 | 98.7 | NO | NO | 130/90 | 110/70 | 140/80 | 130/80 | 140/70 | 140/80 | 140/80 | | 84 | 86 | 82 | 80 | 86 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 56 | 835627 | 20 | 98.8 | NO | NO | 130/80 | 110/70 | 140/80 | 140/80 | 140/90 | 130/80 | 140/80 | | 88 | 86 | 88 | 84 | 82 | 80 | 82 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 57 | 882216 | 20 | 98.6 | NO | NO | 120/80 | 130/90 | 140/80 | 140/80 | 140/90 | 130/80 | 140/80 | | 86 | 88 | 82 | 84 | 80 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 58 | 882798 | 22 | 98.8 | NO | NO | 130/70 | 130/80 | 140/80 | 130/80 | 130/90 | 140/80 | | | 82 | 84 | 82 | 86 | 92 | 94 | | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 59 | 881683 | 22 | 98.7 | NO | NO | 130/80 | 130/70 | 140/80 | 140/80 | 140/90 | 140/80 | | | 84 | 86 | 88 | 92 | 90 | 92 | | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 60 | 882198 | 24 | 98.8 | NO | YES | 130/80 | 110/80 | 140/80 | 140/80 | 140/90 | 130/70 | 140/80 | | 80 | 86 | 82 | 80 | 82 | 84 | | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | NO |
| 61 | 889365 | 22 | 98.7 | NO | YES | 130/70 | 130/70 | 130/82 | 140/90 | 130/80 | 140/80 | | | 84 | 86 | 88 | 80 | 84 | 86 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 62 | 889398 | 22 | 98.6 | YES | NO | 130/80 | 110/70 | 140/84 | 140/90 | 140/100 | 140/90 | 150/90 | | 86 | 84 | 82 | 80 | 84 | 86 | 88 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 63 | 824046 | 20 | 98.8 | NO | NO | 130/90 | 110/70 | 140/90 | 140/100 | 140/90 | 150/100 | 160/100 | | 84 | 86 | 88 | 84 | 82 | 86 | 82 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | YES |
| 64 | 874245 | 22 | 98.7 | NO | YES | 120/80 | 130/80 | 140/90 | 140/90 | 140/80 | 140/100 | 150/90 | 150/100 | 84 | 86 | 80 | 82 | 88 | 84 | 86 | 88 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | 22 | YES |
| 65 | 885338 | 22 | 98.6 | NO | NO | 130/90 | 130/80 | 140/80 | 140/100 | 140/90 | 140/100 | | | 86 | 84 | 80 | 82 | 88 | 94 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | YES |
| 66 | 885502 | 20 | 98.8 | YES | NO | 130/80 | 130/90 | 140/90 | 140/90 | 140/100 | 140/100 | | | 88 | 86 | 80 | 84 | 88 | 92 | | | 20 | 20 | 20 | 20 | 20 | 20 | | | YES |

| Sr. No | MRD NUMBER | Blood Group | Hemogram | Viral Markers | VDRL | FBS | TSH | Urine Routine | NST | IN HYPERTENSIVE PATIENTS_RFT | LFT | Uric Acid | LDH | Serum Proteins | Spot Protein: Creatinine Ratio |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 612695 | 0+ | HB,TLC,PLT-12.3,11.7,264 | NR | NR | 102 | 2.57 | ABNORMAL | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 5.9 | 0.2 |
| 2 | 364555 | A+ | HB,TLC,PLT-10.8,10,166 | NR | NR | 101 | 2.3 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.1 |
| 3 | 364555 | AB+ | HB,TLC,PLT-10.9,20.3,100 | NR | NR | 112 | 3 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | ABNORMA | 6.4 | 0.1 |
| 4 | 716078 | B+ | HB,TLC,PLT-10,13,253 | NR | NR | 98 | 3.5 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.3 | 0.2 |
| 5 | 847414 | AB+ | HB,TLC,PLT-11.5,6.9,207 | NR | NR | 84 | 4.18 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.7 | 0.2 |
| 6 | 713183 | A+ | HB,TLC,PLT-10.6,6.6,233 | NR | NR | 88 | 3.6 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.2 |
| 7 | 802941 | 0+ | HB,TLC,PLT-11.3,7.6,160 | NR | NR | 80 | 3.16 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6 | 0.3 |
| 8 | 848712 | B+ | HB,TLC,PLT-13.3,8.4,164 | NR | NR | 74 | 3.12 | NAD | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.7 | 0.2 |
| 9 | 845714 | AB+ | HB,TLC,PLT-10.6,9.2,236 | NR | NR | 84 | 2.53 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.2 | 0.2 |
| 10 | 820040 | AB+ | HB,TLC,PLT-8,12.3,374 | NR | NR | 80 | 2 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.5 | 0.1 |
| 11 | 837627 | B+ | HB,TLC,PLT-15,10.7,169 | NR | NR | 82 | 2.17 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.8 | 0.2 |
| 12 | 840308 | 0+ | HB,TLC,PLT-13.6,9.5,283 | NR | NR | 111 | 2.35 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.9 |
| 13 | 837803 | 0+ | HB,TLC,PLT-12.1,10.2,178 | NR | NR | 100 | 1.6 | ABNORMAL | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 6.3 | 4.1 |
| 14 | 686287 | 0- | HB,TLC,PLT-10.6,11,269 | NR | NR | 101 | 1.85 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.5 | 0.1 |
| 15 | 837533 | AB+ | HB,TLC,PLT-8.3,11,268 | NR | NR | 90 | 3.8 | NAD | NON REASURRING | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.8 | 0.32 |
| 16 | 602785 | B+ | HB,TLC,PLT-9.9,8.3,172 | NR | NR | 137 | 2.66 | ABNORMAL | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 5.3 | 0.4 |
| 17 | 716186 | B+ | HB,TLC,PLT-10,12.2,256 | NR | NR | 86 |  | NAD | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 6.6 | 0.2 |
| 18 | 714540 | B+ | HB,TLC,PLT-10.5,12.3,230 | NR | NR | 90 | 2.3 | ABNORMAL | NON REASURRING | NORMAL | NORMAL | ABNORMA | ABNORMA | 5.9 | 3 |
| 19 | 120801 | B+ | HB,TLC,PLT-12.8,11.6,336 | NR | NR | 118 | 4.24 | ABNORMAL | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.2 |
| 20 | 640008 | A- | HB,TLC,PLT-8.4,7.1,333 | NR | NR | 84 | 1.92 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.3 |
| 21 | 713018 | AB+ | HB,TLC,PLT-11.1,13.2,246 | NR | NR | 90 | 2.8 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | NORMAL | ABNORMA | 6.2 | 0.3 |
| 22 | 7921 | B+ | HB,TLC,PLT-11.6,11.1,265 | NR | NR | 81 | 2.6 | ABNORMAL | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 5.9 | 0.2 |
| 23 | 705147 | B+ | HB,TLC,PLT-13.7,20.7,236 | NR | NR | 94 | 1.33 | ABNORMAL | NON REASURRING | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 4.3 | 13 |
| 24 | 565864 | AB+ | HB.TLC,PLT-12.6,7.7,160 | NR | NR | 88 | 2.8 | ABNORMAL | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6 | 0.1 |
| 25 | 708573 | O+ | HB,TLC,PLT-9.1,15.6,263 | NR | NR | 105 | 3.65 | NAD | NON REASURRING | ABNORMAL | NORMAL | NORMAL | ABNORMA | 6.8 | 0.3 |
| 26 | 704144 | B+ | HB,TLC,PLT-9.8,8.5,256 | NR | NR | 97 | 1.2 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.2 |
| 27 | 571394 | B+ | HB,TLC,PLT-10.7,10.5,137 | NR | NR | 120 | 1.29 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 28 | 661119 | A+ | HB,TLC,PLT-10.6,10.8,447 | NR | NR | 117 | 2.27 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 7.3 | 0.2 |
| 29 | 541325 | B+ | HB,TLC,PLT-10.9,13.4,275 | NR | NR | 102 | 2.04 | ABNORMAL | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.1 |
| 30 | 800260 | B+ | HB,TLC,PLT-11.9,8.6,176 | NR | NR | 88 | 4.76 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.2 | 0.4 |
| 31 | 801458 | A+ | HB,TLC,PLT-12.3,20.2,319 | NR | NR | 77 | 3.24 | NAD | REACTIVE | NORMAL | ABNORMAL | NORMAL | NORMAL | 6 | 0.2 |
| 32 | 792594 | AB+ | HB,TLC,PLT-12.3,13.7,326 | NR | NR | 80 | 2.84 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.4 | 0.2 |
| 33 | 750480 | B- | HB,TLC,PLT-15.7,17,100 | NR | NR | 93 | 1.5 | ABNORMAL | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.1 | 0.8 |
| 34 | 835322 | B- | HB,TLC,PLT-12.2,17.4,180 | NR | NR | 86 | 1.22 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.5 | 0.1 |
| 35 | 836097 | O+ | HB,TLC,PLT-12,22.1,199 | NR | NR | 84 | 0.56 | NAD | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 5.9 | 0.4 |
| 36 | 832838 | O+ | HB,TLC,PLT-11.9,7.6,280 | NR | NR | 79 | 4.69 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.2 |
| 37 | 484927 | O- | HB,TLC,PLT-11.6,9.8,244 | NR | NR | 109 | 2.69 | NAD | REACTIVE | NORMAL | ABNORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 38 | 858044 | AB+ | HB,TLC,PLT-10.7,10.3,97 | NR | NR | 128 | 1.8 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.3 |
| 39 | 450551 | B- | HB,TLC,PLT-7.3,11.1,503 | NR | NR | 107 | 3.15 | NAD | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 6 | 0.2 |
| 40 | 859391 | B+ | HB,TLC,PLT-11.6,20.7,236 | NR | NR | 113 | 2.18 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.3 | 0.3 |
| 41 | 618016 | O+ | HB,TLC,PLT-7.1,11.2,150 | NR | NR | 86 | 3.12 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 42 | 711775 | O+ | HB,TLC,PLT-11.2,11.6.168 | NR | NR | 82 | 2.6 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.3 |
| 43 | 857581 | B+ | HB,TLC,PLT-9.2,13.6,140 | NR | NR | 102 | 3.76 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.8 | 0.2 |
| 44 | 850065 | B+ | HB,TLC,PLT-8.9,10.9,210 | NR | NR | 88 | 1.41 | ABNORMAL | REACTIVE | ABNORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.3 |
| 45 | 775214 | B+ | HB,TLC,PLT-12.3,9.7,154 | NR | NR | 90 | 2.8 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.7 | 0.2 |
| 46 | 866673 | B+ | HB,TLC,PLT-10,12,250 | NR | NR | 98 | 1.1 | NAD | REACTIVE | NORMAL | ABNORMAL | NORMAL | NORMAL | 5.7 | 0.5 |
| 47 | 172189 | A+ | HB,TLC,PLT-11.3,9.6,157 | NR | NR | 81 | 1.8 | NAD | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 7.2 | 0.4 |
| 48 | 770592 | B+ | HB,TLC,PLT-13.8,9.7,100 | NR | NR | 98 | 1.8 | NAD | REACTIVE | ABNORMAL | ABNORMAL | NORMAL | NORMAL | 6.5 | 0.3 |
| 49 | 718218 | O+ | HB,TLC,PLT-11.5,10.3,218 | NR | NR | 90 | 2.5 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 50 | 796664 | B+ | HB,TLC,PLT-11.9,12.8,362 | NR | NR | 92 | 2 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6 | 0.3 |
| 51 | 729147 | B+ | HB,TLC,PLT-10,13,252 | NR | NR | 87 | 3.05 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.2 |
| 52 | 882761 | A1B+ | HB,TLC,PLT-10.8,8,167 | NR | NR | 80 | 3.8 | NAD | REACTIVE | NORMAL | ABNORMAL | NORMAL | NORMAL | 6.3 | 0.2 |
| 53 | 869544 | B+ | HB,TLC,PLT-14.2,11.9,180 | NR | NR | 83 | 1.6 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 54 | 881584 | B+ | HB,TLC,PLT-9.8,14.3,180 | NR | NR | 72 | 3.17 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.2 |
| 55 | 882183 | B+ | HB,TLC,PLT-11.6,9.6,93 | NR | NR | 90 | 0.63 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.3 |
| 56 | 835627 | O+ | HB,TLC,PLT-9.3,8.6,158 | NR | NR | 88 | 2.29 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.2 |
| 57 | 882216 | B- | HB,TLC,PLT-10.8,9.1,257 | NR | NR | 90 | 2.8 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 5.8 | 0.3 |
| 58 | 882798 | B+ | HB,TLC,PLT-10.8,9.1,187 | NR | NR | 83 | 1.6 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 59 | 881683 | AB+ | HB,TLC,PLT-10.7,12.5,178 | NR | NR | 86 | 2.35 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 5.8 | 0.3 |
| 60 | 882198 | B+ | HB,TLC,PLT-13.1,13.4,190 | NR | NR | 80 | 2.5 | NAD | NON REASURRING | NORMAL | NORMAL | NORMAL | NORMAL | 6 | 0.2 |
| 61 | 889365 | O+ | HB,TLC,PLT-12.5,10.1,191 | NR | NR | 98 | 2.31 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.3 |
| 62 | 889398 | O+ | HB,TLC,PLT-13.5,8.6,239 | NR | NR | 90 | 4.3 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.3 | 1.2 |
| 63 | 824046 | A+ | HB,TLC,PLT-11.7,10,130 | NR | NR | 84 | 3.2 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | ABNORMA | 6.8 | 0.4 |
| 64 | 874245 | B+ | HB,TLC,PLT-11.7,8.2,63 | NR | NR | 88 | 1.8 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | NORMAL | NORMAL | 6.5 | 0.2 |
| 65 | 885338 | A+ | HB,TLC,PLT-12.9,13.5,368 | NR | NR | 94 | 0.6 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 66 | 885502 | B+ | HB,TLC,PLT-11,12,152 | NR | NR | 89 | 2.5 | NAD | REACTIVE | NORMAL | ABNORMAL | NORMAL | NORMAL | 5.8 | 0.2 |

| Sr. No | MRD NUMBER | Fundus Examination | Dose of Antihypertensi | Route | Frequency | Duration (Months) | DEVELPOMENT OF HYPERTENSION | Date of birth | Time of birth | Sex | Weight | APGAR Score | NICU Admission | NEONATAL DEATH | comobidity |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 612695 | NORMAL | 100MG | P/0 | TDS | 3 | YES | 8/1/2019 | 10pm | M | 2.8 | 8 | No | No | - |
| 2 | 364555 | NORMAL | 100MG | P/0 | BD | 3 | YES | 12/1/2019 | 8:07pm | M | 2.62 | 7 | No | No | - |
| 3 | 364555 | NORMAL | 200MG | P/0 | TDS | 3 | YES | 18/1/2019 | 4:00pm | F | 3260gm | 9 | No | No | - |
| 4 | 716078 | MILD HTN RETINOPATHY | 200MG | P/0 | BD | 3 | YES | 28/1/2019 | 5:43pm | M | 880gm | 4 | Yes | Yes | PRETERM |
| 5 | 847414 | NORMAL | 100MG | P/0 | BD | 4 | YES | 1/2/2019 | 12:26pm | M | 2.24 | 6 | Yes | No | PRETERM |
| 6 | 713183 | NORMAL | 200MG | P/0 | BD | 3 | YES | 3/2/2019 | 11:10pm | M | 4.02 | 8 | No | No | - |
| 7 | 802941 | NORMAL | 200MG | P/0 | BD | 3 | YES | 5/2/2019 | 2:30pm | F | 2.76 | 8 | No | No | - |
| 8 | 848712 | NORMAL | 200MG | P/O | BD | 3 | YES | 10/2/2019 | 12:54pm | F | 1840gm | 5 | Yes | No | HYPOGLYCEMIA |
| 9 | 845714 | NORMAL | 100MG | P/O | BD | 3 | YES | 15/2/2019 | 10:48am | F | 2040g | 5 | Yes | Yes | RDS |
| 10 | 820040 | NORMAL | 200MG | P/0 | BD | 4 | YES | 19/2/2019 | 8:22am | F | 3.36 | 8 | No | No | - |
| 11 | 837627 | NORMAL | 200MG | P/O | TDS | 3 | YES | 26/2/2019 | 10:04am | M | 3.48 | 6 | Yes | No | MAS |
| 12 | 840308 | NORMAL | 200MG | P/O | TDS | 3 | YES | 1/3/2019 | 1:36am | F | 2.7 | 8 | No | No | - |
| 13 | 837803 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 3 | YES | 4/3/2019 | 12pm | M | 3 | 7 | Yes | No | RDS |
| 14 | 686287 | NORMAL | 200MG | P/O | TDS | 3 | YES | 6/3/2019 | 11:21am | M | 3.94 | 9 | No | No | - |
| 15 | 837533 | NORMAL | 200MG | P/O | TDS | 3 | YES | 10/3/2019 | 2pm | M | 3.09 | 10 | No | No | - |
| 16 | 602785 | NORMAL | 200MG | P/O | BD | 3 | YES | 14/3/2019 | 11:25am | F | 2.24 | 6 | Yes | No | - |
| 17 | 716186 | NORMAL | 200MG | P/O | TDS | 3 | YES | 18/3/2019 | 10:27am | M | 2.64 | 6 | Yes | No | RDS |
| 18 | 714540 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 3 | YES | 20/3/2019 | 12:26pm | M | 2.82 | 9 | No | No | PRETERM |
| 19 | 120801 | NORMAL | 100MG | P/O | TDS | 3 | YES | 22/3/2019 | 10:26am | M | 2.12 | 6 | Yes | No | PRETERM |
| 20 | 640008 | NORMAL | 200MG | P/O | TDS | 4 | YES | 26/3/2019 | 9:29am | M | 2.7 | 9 | No | No | RDS |
| 21 | 713018 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 3 | YES | 28/3/2019 | 8:14am | F | 1.44 | 4 | Yes | No | PRETERM |
| 22 | 7921 | NORMAL | 200MG | P/O | BD | 2 | YES | 1/4/2019 | 9am | F | 2 | 6 | Yes | No | PRETERM |
| 23 | 705147 | NORMAL | 100MG | P/O | BD | 3 | YES | 9/4/2019 | 2:07pm | M | 1.53 | 4 | Yes | No | PRETERM |
| 24 | 565864 | NORMAL | 100MG | P/0 | BD | 3 | YES | 11/4/2019 | 12:26pm | M | 2.3 | 6 | Yes | No | PRETERM |
| 25 | 708573 | NORMAL | 200MG | P/O | TDS | 3 | YES | 15/4/2019 | 4:29pm | F | 1.16 | 4 | Yes | No | - |
| 26 | 704144 | NORMAL | 200MG | P/O | TDS | 3 | YES | 19/4/2019 | 11:02pm | M | 2 | 6 | Yes | No | - |
| 27 | 571394 | NORMAL | 200MG | P/O | TDS | 3 | YES | 23/4/2019 | 1pm | F | 3.4 | 8 | No | No | - |
| 28 | 661119 | NORMAL | 100MG | P/O | BD | 3 | YES | 26/4/2019 | 1:19pm | M | 3.32 | 7 | Yes | No | RDS |
| 29 | 541325 | NORMAL | 200MG | P/O | TDS | 4 | YES | 30/4/2019 | 8:38pm | F | 3.4 | 9 | No | No | - |
| 30 | 800260 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 3 | YES | 2/5/2019 | 4pm | M | 3.8 | 7 | Yes | No | RDS |
| 31 | 801458 | NORMAL | 100MG | P/O | BD | 3 | YES | 8/5/2019 | 9:40am | F | 2.82 | 7 | Yes | No | MAS |
| 32 | 792594 | NORMAL | 200MG | P/O | TDS | 3 | YES | 10/5/2019 | 12pm | F | 3.4 | 7 | Yes | No | - |
| 33 | 750480 | NORMAL | 200MG | P/O | TDS | 3 | YES | 16/5/2019 | 9:17am | F | 1860gm | 4 | Yes | No | - |
| 34 | 835322 | NORMAL | 200MG | P/O | TDS | 3 | YES | 20/5/2019 | 3:57pm,3:5 | F | 2.8 | 7 | No | No | - |
| 35 | 836097 | NORMAL | 200MG | P/O | TDS | 3 | YES | 29/5/19 | 8:44pm | M | 2100gm | 6 | Yes | No | PRETERM |
| 36 | 832838 | NORMAL | 100MG | P/O | TDS | 3 | YES | 1/6/2019 | 9:58am | M | 2580gm | 7 | No | No | RDS |
| 37 | 484927 | NORMAL | 100MG | P/O | TDS | 3 | YES | 5/6/2019 | 7:19pm | M | 2700gm | 6 | Yes | No | MAS |
| 38 | 858044 | NORMAL | 200MG | P/O | TDS | 3 | YES | 7/6/2019 | 12:02pm | M | 3.48 | 9 | No | No | - |
| 39 | 450551 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 3 | YES | 9/6/2019 | 8:19pm | F | 2.82 | 7 | Yes | No | RDS |
| 40 | 859391 | NORMAL | 100MG | P/O | TDS | 4 | YES | 12/6/2019 | 9:18am | F | 2.8 | 7 | Yes | No | MAS |
| 41 | 618016 | NORMAL | 200MG | P/O | TDS | 3 | YES | 20/6/2019 | 6:47pm | F | 2460gm | 6 | Yes | No | RDS |
| 42 | 711775 | NORMAL | 100MG | P/O | BD | 4 | YES | 28/6/2019 | 3:22pm | F | 3.6 | 9 | No | No | - |
| 43 | 857581 | NORMAL | 200MG | P/O | BD | 2 | YES | 9/7/2019 | 10:56pm | M | 1.96 | 4 | Yes | No | PRETERM |
| 44 | 850065 | NORMAL | 100MG | P/O | TDS | 3 | YES | 20/7/2019 | 12pm | M | 2 | 5 | Yes | No | PRETERM |
| 45 | 775214 | NORMAL | 200MG | P/O | TDS | 4 | YES | 29/7/2019 | 7:53pm | M | 2940gm | 8 | No | No | - |
| 46 | 866673 | NORMAL | 100MG | P/O | TDS | 3 | YES | 19/8/2019 | 1:07pm | F | 3260gm | 9 | No | No | - |
| 47 | 172189 | NORMAL | 100MG | P/O | TDS | 3 | YES | 22/8/2019 | 2:30pm | M | 3290gm | 10 | No | No | - |
| 48 | 770592 | NORMAL | 200MG | P/O | TDS | 3 | YES | 26/8/2019 | 7:20am | M | 3.06 | 7 | Yes | No | - |
| 49 | 718218 | NORMAL | 100MG | P/O | TDS | 3 | YES | 28/8/2019 | 9:17am | M | 3 | 8 | No | No | - |
| 50 | 796664 | NORMAL | 200MG | P/O | BD | 4 | YES | 29/8/2019 | 12:02pm | F | 3.4 | 9 | No | No | - |
| 51 | 729147 | NORMAL | 100MG | P/O | BD | 4 | YES | 30/8/2019 | 4:02pm | F | 4 | 10 | No | No | - |
| 52 | 882761 | NORMAL | 100MG | P/O | TDS | 3 | YES | 4/9/2019 | 11:35am | M | 2380g | 7 | Yes | No | PRETERM |
| 53 | 869544 | NORMAL | 200MG | P/O | BD | 4 | YES | 7/9/2019 | 3:31am | M | 2.88 | 8 | No | No | - |
| 54 | 881584 | NORMAL | 100MG | P/O | TDS | 3 | YES | 12/9/2019 | 10:21am | M | 2.8 | 9 | No | No | - |
| 55 | 882183 | NORMAL | 200MG | P/O | BD | 3 | YES | 15/9/2019 | 9:54pm | M | 2.64 | 7 | Yes | No | - |
| 56 | 835627 | NORMAL | 100MG | P/O | TDS | 3 | YES | 20/9/2019 | 11:49am | M | 3.2 | 8 | No | No | - |
| 57 | 882216 | NORMAL | 200MG | P/O | BD | 3 | YES | 21/9/2019 | 6:25pm | F | 3.1 | 9 | No | No | - |
| 58 | 882798 | NORMAL | 100MG | P/O | BD | 3 | YES | 24/9/2019 | 1:47pm | F | 1735g | 5 | Yes | No | - |
| 59 | 881683 | NORMAL | 200MG | P/O | TDS | 4 | YES | 26/9/2019 | 2:00pm | M | 1.96 | 5 | Yes | No | PRETERM |
| 60 | 882198 | NORMAL | 200MG | P/O | BD | 3 | YES | 29/9/2019 | 4:40am | F | 2.4 | 6 | Yes | No | RDS |
| 61 | 889365 | NORMAL | 200MG | P/O | TDS | 3 | YES | 30/9/2019 | 11:35am | F | 1.78 | 4 | Yes | No | PRETERM |
| 62 | 889398 | MILD HTN RETINOPATHY | 100MG | P/O | BD | 3 | YES | 5/10/2019 | 12:03pm | F | 1.64 | 5 | Yes | No | PRETERM |
| 63 | 824046 | NORMAL | 100MG | P/O | TDS | 3 | YES | 9/10/2019 | 11:02pm | M | 2.82 | 9 | No | No | PRETERM |
| 64 | 874245 | NORMAL | 200MG | P/O | TDS | 3 | YES | 10/10/2019 | 11:37am | F | 2.93 | 7 | Yes | No | - |
| 65 | 885338 | NORMAL | 100MG | P/O | BD | 3 | YES | 12/10/2019 | 12:04pm | M | 2.74 | 9 | No | No | - |
| 66 | 885502 | NORMAL | 200MG | P/O | TDS | 3 | YES | 15/10/2019 | 2:11am | F | 1.96 | 6 | Yes | No | - |

| Sr. No | MRD NUMBER | Initials | AGE | Weight | Height | BMI | Area | DATE OF ADMISSION | ADMISSION NUMBER | EDUCATION | OCCUPATION | PARITY | PARITY | BOOKED/UNBOOKED | POG | LOCATION OF PLACENTA ON ULTRASOUND |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | 791980 | S | 28 | 77 | 5'4'' | 26.5 | URBAN | 8/10/2019 | 2020008470 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 21WEEKS | LATERAL |
| 68 | 891080 | N | 28 | 67 | 5' | 24.2 | URBAN | 9/10/2019 | 2020007738 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4P2L1A1 | YES | 20WEEKS | LATERAL |
| 69 | 890631 | B | 29 | 82.5 | 5'1'' | 30.2 | RURAL | 12/10/2019 | 2020007426 | GRADUATE | STAFF NURSE | MULTIGRAVIDA | G3A2 | YES | 22WEEKS | POSTERIOR |
| 70 | 888167 | RK | 32 | 80 | 5' | 30.1 | URBAN | 15/10/2019 | 2020006414 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 19WEEKS | POSTERIOR |
| 71 | 894138 | KK | 22 | 77 | 5'3'' | 26.1 | URBAN | 17/10/2019 | 2020008933 | GRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 72 | 891992 | AB | 25 | 68 | 5'3'' | 23.4 | URBAN | 18/10/2019 | 2020008049 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 18WEEKS | POSTERIOR |
| 73 | 558647 | R | 30 | 72 | 5'1'' | 27.5 | URBAN | 20/10/2019 | 2020008718 | POSTGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 22WEEKS | LATERAL |
| 74 | 117378 | VK | 35 | 105 | 5'2'' | 35.9 | URBAN | 24/10/2019 | 2020008504 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3P1L1A1 | YES | 20WEEKS | LATERAL |
| 75 | 993566 | R | 34 | 80 | 5' | 28.2 | RURAL | 27/10/2019 | 2020009462 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G4P3L2 | YES | 21WEEK | LATERAL |
| 76 | 893508 | LK | 29 | 50 | 5'4'' | 27.4 | URBAN | 28/10/2019 | 2020008694 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 19WEEKS | LATERAL |
| 77 | 28511 | S | 26 | 55 | 5' | 18.9 | URBAN | 30/10/2019 | 2020008655 | POSTGRADUATE | TEACHER | MULTIGRAVIDA | G2P1L1 | NO | 20WEEKS | LATERAL |
| 78 | 892668 | D | 25 | 80 | 5'2'' | 32.3 | URBAN | 2/11/2019 | 2020008364 | POSTGRADUATE | TEACHER | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |
| 79 | 71774 | S | 25 | 61 | 5'' | 23.7 | URBAN | 3/11/2019 | 2020009219 | GRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 18WEEKS | LATERAL |
| 80 | 908477 | LR | 25 | 78 | 5'2'' | 26.5 | URBAN | 6/11/2019 | 2020015047 | GRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 81 | 908501 | MS | 29 | 84 | 5'4'' | 24.6 | RURAL | 8/11/2019 | 2020015063 | GRADUATE | LAB.TECHNICIAN | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |
| 82 | 908761 | IK | 27 | 69 | 5'4'' | 22.7 | RURAL | 10/11/2019 | 2020015512 | GRADUATE | STAFF NURSE | PRIMIGRAVIDA | G1 | YES | 18WEEKS | ANTERIOR |
| 83 | 911028 | S | 32 | 53 | 5'2'' | 20.2 | URBAN | 13/11/2019 | 2020016204 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | POSTERIOR |
| 84 | 910716 | SK | 36 | 64 | 5'1'' | 24.2 | URBAN | 16/11/2019 | 2020016101 | UNDERGRADUATE | PHARMACIST | MULTIGRAVIDA | G2P1L1 | YES | 22WEEKS | LATERAL |
| 85 | 780034 | N | 30 | 62 | 4'9'' | 25 | URBAN | 18/11/2019 | 2020016107 | POSTGRADUATE | TEACHER | MULTIGRAVIDA | G3A2 | YES | 20WEEKS | LATERAL |
| 86 | 823208 | R | 28 | 67 | 5'1'' | 23.4 | URBAN | 21/11/2019 | 2020028162 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |
| 87 | 878277 | A | 24 | 57 | 5'4'' | 20.8 | URBAN | 23/11/2019 | 2020028857 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L0 | YES | 20WEEKS | LATERAL |
| 88 | 933090 | Y | 31 | 78 | 5'2'' | 27.3 | URBAN | 26/11/2019 | 2020029799 | GRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L0 | YES | 18WEEKS | LATERAL |
| 89 | 929328 | R | 27 | 70 | 5' | 30.1 | RURAL | 28/11/2019 | 2020028140 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 90 | 927480 | B | 30 | 60 | 5'2'' | 24.2 | URBAN | 30/11/2019 | 2020026719 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3P1LOA1 | YES | 22WEEKS | LATERAL |
| 91 | 929328 | R | 27 | 70G | 5' | 25.8 | RURAL | 2/12/2019 | 2020028140 | UNDERGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 92 | 944313 | JK | 36 | 64 | 5'3'' | 25 | RURAL | 4/12/2019 | 2020035859 | GRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3P2L2 | YES | 22WEEKS | LATERAL |
| 93 | 943817 | PK | 45 | 72 | 5'2'' | 28.2 | RURAL | 6/12/2019 | 2020035297 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 18WEEKS | LATERAL |
| 94 | 934080 | M | 26 | 62 | 5'4'' | 21.9 | RURAL | 10/12/2019 | 2020035298 | POSTGRADUATE | HOUSEWIFE | PRIMIGRAVIDA | G1 | YES | 20WEEKS | LATERAL |
| 95 | 678459 | RB | 25 | 64 | 5'1'' | 25.8 | URBAN | 13/12/2019 | 2020035059 | POSTGRADUATE | TEACHER | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |
| 96 | 945451 | SK | 40 | 56 | 5'3'' | 19.5 | RURAL | 15/12/2019 | 2020035992 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G3P2L2 | YES | 24WEEKS | ANTERIOR |
| 97 | 813732 | PR | 31 | 81 | 5'4'' | 30.5 | URBAN | 17/12/2019 | 2020036007 | GRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 20WEEKS | POSTERIOR |
| 98 | 882380 | KK | 39 | 76 | 5'2'' | 24.2 | RURAL | 21/12/2019 | 2020036036 | POSTGRADUATE | TEACHER | MULTIGRAVIDA | G2P1L1 | YES | 22WEEKS | LATERAL |
| 99 | 303888 | MM | 31 | 78 | 5'3'' | 26.5 | URBAN | 22/12/2019 | 2020036021 | UNDERGRADUATE | HOUSEWIFE | MULTIGRAVIDA | G2P1L1 | YES | 24WEEKS | LATERAL |
| 100 | 894637 | PK | 24 | 72 | 5'1'' | 24.2 | URBAN | 26/12/2019 | 2020035623 | GRADUATE | TEACHER | PRIMIGRAVIDA | G1 | YES | 22WEEKS | LATERAL |

| Sr. No | MRD NUMBER | CALCIFICATION OF PLACENTA ON ULTRASOUND | ULTRASOUND FOR LOCATION OF PLACENTA | PAST HISTORY | FAMILY HISTORY | SOCIO-ECONOMIC HISTORY | SOCIOECONOMIC STATUS | NUTRITIONAL HISTORY | PERIOD OF GESTATION AT THE TIME OF DELIVERY |
|---|---|---|---|---|---|---|---|---|---|
| 67 | 791980 | NO | 21WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 37+4WEEKS |
| 68 | 891080 | NO | 20WEEKS | HTN | HTN | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 33+2WEEKS |
| 69 | 890631 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 37WEEKS |
| 70 | 888167 | NO | 19WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 37+3WEEKS |
| 71 | 894138 | NO | 20WEEKS | HTN | HTN | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 38+6WEEKS |
| 72 | 891992 | NO | 18WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 36+3WEEKS |
| 73 | 558647 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 36WEEKS |
| 74 | 117378 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 40+2WEEKS |
| 75 | 993566 | NO | 21WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 38+2WEEKS |
| 76 | 893508 | NO | 19WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 34+6WEEKS |
| 77 | 28511 | NO | 2OWEEKS | HTN | HTN | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 39+1WEEK |
| 78 | 892668 | NO | 22WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 35WEEKS |
| 79 | 71774 | NO | 18WEEKS | HTN | HTN | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 36+3WEEKS |
| 80 | 908477 | NO | 20WEEKS | HTN | HTN | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 29+4WEEKS |
| 81 | 908501 | NO | 22WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 32+5WEEKS |
| 82 | 908761 | NO | 18WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 37WEEKS |
| 83 | 911028 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 34+4WEEKS |
| 84 | 910716 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 34+5WEEKS |
| 85 | 780034 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 39WEEKS |
| 86 | 823208 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 38+3WEEKS |
| 87 | 878277 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 33+1WEEK |
| 88 | 933090 | NO | 18 WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 32WEEKS |
| 89 | 929328 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 35+4WEEKS |
| 90 | 927480 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 32+5WEEKS |
| 91 | 929328 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 35+4WEEKS |
| 92 | 944313 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 32+5WEEKS |
| 93 | 943817 | NO | 18WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 35+1WEEK |
| 94 | 934080 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER CLASS | ADEQUATE | 36WEEKS |
| 95 | 678459 | NO | 22WEEKS | HTN | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 35WEEKS |
| 96 | 945451 | NO | 24WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER CLASS | ADEQUATE | 36WEEKS |
| 97 | 813732 | NO | 20WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 36+2WEEKS |
| 98 | 882380 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | LOWER MIDDLE CLASS | ADEQUATE | 36WEEKS |
| 99 | 303888 | NO | 24WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER LOWER CLASS | ADEQUATE | 38+1WEEK |
| 100 | 894637 | NO | 22WEEKS | NO RELEVANT | NO SIGNIFICANT | ADEQUATE | UPPER MIDDLE CLASS | ADEQUATE | 37+5WEEKS |

| Sr. No | MRD NUMBER | PRETERM / TERM | MODE OF DELIVERY | VAGINAL (WITH FORCEPS/WITHOUT FORCEPS CAESAREAN | MATERNAL COMPLICATIONS_VOMITING | HEADACHE | BLURRING OF VISION | EPIGASTRIC PAIN | PULSE | SBP | DBP |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | 791980 | TERM | LSCS | CAESSAREAN | YES | YES | NO | YES | 88 | 150 | 100 |
| 68 | 891080 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | YES | NO | 84 | 140 | 80 |
| 69 | 890631 | TERM | NVD | NVD WITH RMLE WITH FORCEPS | NO | NO | NO | NO | 86 | 140 | 90 |
| 70 | 888167 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 90 | 150 | 90 |
| 71 | 894138 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 92 | 140 | 90 |
| 72 | 891992 | LATE PRETERM | NVD | NVD WITH RMLE WITH FORCEPS | NO | NO | NO | NO | 84 | 150 | 90 |
| 73 | 558647 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 90 |
| 74 | 117378 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 82 | 140 | 90 |
| 75 | 993566 | TERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 86 | 150 | 90 |
| 76 | 893508 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 84 | 140 | 90 |
| 77 | 28511 | TERM | LSCS | CAESSAREAN | NO | NO | YES | NO | 89 | 140 | 90 |
| 78 | 892668 | LATE PRETERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 90 | 140 | 90 |
| 79 | 71774 | EARLY PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 82 | 150 | 90 |
| 80 | 908477 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | YES | NO | 84 | 150 | 90 |
| 81 | 908501 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | YES | NO | 88 | 140 | 90 |
| 82 | 908761 | TERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 86 | 140 | 90 |
| 83 | 911028 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | YES | 84 | 150 | 110 |
| 84 | 910716 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 89 | 160 | 110 |
| 85 | 780034 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 98 | 140 | 90 |
| 86 | 823208 | TERM | LSCS | CAESSAREAN | NO | NO | NO | NO | 84 | 140 | 90 |
| 87 | 878277 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 150 | 90 |
| 88 | 933090 | EARLY PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 140 | 90 |
| 89 | 929328 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 92 | 140 | 90 |
| 90 | 927480 | EARLY PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 98 | 140 | 90 |
| 91 | 929328 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 92 | 140 | 90 |
| 92 | 944313 | EARLY PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 84 | 150 | 90 |
| 93 | 943817 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 88 | 140 | 90 |
| 94 | 934080 | LATE PRETERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 84 | 140 | 90 |
| 95 | 678459 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 140 | 90 |
| 96 | 945451 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 98 | 150 | 90 |
| 97 | 813732 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 86 | 140 | 90 |
| 98 | 882380 | LATE PRETERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 84 | 140 | 90 |
| 99 | 303888 | TERM | LSCS | CAESSAREAN | YES | NO | NO | NO | 89 | 140 | 90 |
| 100 | 894637 | TERM | LSCS | CAESSAREAN | YES | YES | NO | NO | 88 | 150 | 90 |

| Sr. No | MRD NUMBER | RESPIRATORY RATE | TEMPERATURE | EDEMA_GENERALISED | EDEMA_PEDAL | SBP_18HRS | SBP_20HRS | SBP_22HRS | SBP_24HRS | SBP_28HRS | SBP_32HRS | SBP_36HRS | SBP_40HRS | PULSE_18HRS | PULSE_20 | PULSE_22 | PULSE_24 | PULSE_28 | PULSE_32 | PULSE_36 | PULSE | RR_18HRS | RR_20 | RR_22 | RR_24 | RR_28 | RR_32 | RR_36 | RR_40 | HTN |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | 791980 | 22 | 98.6 | NO | YES | 120/80 | 130/80 | 140/80 | 140/80 | 140/90 | 130/80 | 140/90 | | 84 | 88 | 86 | 80 | 82 | 88 | 86 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 68 | 891080 | 22 | 98.5 | YES | NO | 130/70 | 110/70 | 130/80 | 140/90 | 150/100 | 140/90 | 140/81 | | 84 | 88 | 86 | 82 | 88 | 90 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | YES |
| 69 | 890631 | 24 | 98.7 | NO | NO | 130/80 | 130/90 | 140/80 | 140/90 | 140/90 | 150/100 | 140/90 | | 94 | 92 | 90 | 92 | 94 | 96 | 94 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | YES |
| 70 | 888167 | 22 | 98.8 | NO | YES | 120/80 | 130/80 | 140/80 | 140/88 | 140/100 | 140/90 | 150/100 | | 80 | 86 | 84 | 82 | 88 | 84 | 82 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 71 | 894138 | 20 | 98.7 | NO | YES | 130/80 | 110/70 | 140/90 | 140/100 | 140/90 | 140/90 | 140/100 | | 84 | 86 | 88 | 82 | 84 | 80 | 84 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | YES |
| 72 | 891992 | 22 | 98.6 | NO | NO | 130/70 | 110/70 | 140/80 | 140/80 | 140/90 | 140/90 | 150/100 | | 82 | 84 | 88 | 80 | 86 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | YES |
| 73 | 558647 | 22 | 98.8 | NO | NO | 130/80 | 130/90 | 140/80 | 140/70 | 140/90 | 140/80 | 130/90 | | 84 | 86 | 80 | 82 | 84 | 80 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 74 | 117378 | 20 | 98.7 | NO | NO | 130/70 | 130/90 | 140/90 | 140/80 | 130/70 | 140/90 | 130/90 | 140/80 | 89 | 86 | 88 | 80 | 82 | 84 | 86 | 88 | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 75 | 993566 | 22 | 98.8 | NO | NO | 130/70 | 130/80 | 140/80 | 140/90 | 140/80 | 140/80 | 140/90 | | 86 | 84 | 82 | 84 | 86 | 82 | 84 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 76 | 893508 | 22 | 98.6 | YES | NO | 130/80 | 130/70 | 140/90 | 140/90 | 140/100 | 150/100 | | | 84 | 80 | 86 | 88 | 82 | 84 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | YES |
| 77 | 28511 | 24 | 98.8 | YES | YES | 130/80 | 120/80 | 140/98 | 140/100 | 140/100 | 140/110 | 140/90 | | 74 | 72 | 76 | 70 | 74 | 76 | 78 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | YES |
| 78 | 892668 | 22 | 98.7 | NO | NO | 130/80 | 110/70 | 140/90 | 140/90 | 140/90 | 140/110 | | | 84 | 82 | 86 | 88 | 84 | 82 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | YES |
| 79 | 71774 | 18 | 98.6 | NO | YES | 130/70 | 120/80 | 140/80 | 140/100 | 140/90 | 140/90 | 140/94 | | 80 | 82 | 84 | 80 | 86 | 84 | 82 | | 18 | 18 | 18 | 18 | 18 | 18 | 18 | | YES |
| 80 | 908477 | 22 | 99.8 | YES | YES | 130/90 | 110/70 | 140/80 | 140/100 | 150/110 | | | | 94 | 96 | 98 | 98 | 96 | | | | 22 | 22 | 22 | 22 | 22 | | | | YES |
| 81 | 908501 | 22 | 98.8 | YES | NO | 130/80 | 130/80 | 140/90 | 140/100 | 140/90 | 150/110 | | | 84 | 86 | 88 | 90 | 92 | 94 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | YES |
| 82 | 908761 | 20 | 98.7 | NO | YES | 130/80 | 130/90 | 140/80 | 140/80 | 140/90 | 130/80 | 140/90 | | 82 | 84 | 86 | 88 | 84 | 82 | 84 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | YES |
| 83 | 911028 | 22 | 98.6 | NO | NO | 130/70 | 120/80 | 140/80 | 140/100 | 140/90 | 180/110 | | | 84 | 88 | 86 | 90 | 92 | 94 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | YES |
| 84 | 910716 | 22 | 98.8 | NO | NO | 130/80 | 130/80 | 130/80 | 140/80 | 130/80 | 140/90 | | | 89 | 92 | 90 | 92 | 96 | 98 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 85 | 780034 | 24 | 98.6 | NO | NO | 130/80 | 120/80 | 140/70 | 140/80 | 140/90 | 130/90 | 130/80 | | 84 | 82 | 86 | 88 | 80 | 82 | 84 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | NO |
| 86 | 823208 | 20 | 98.8 | NO | NO | 130/80 | 120/80 | 130/80 | 140/80 | 140/90 | 140/80 | 140/80 | | 80 | 84 | 80 | 84 | 86 | 88 | 90 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 87 | 878277 | 22 | 98.6 | NO | NO | 130/70 | 130/80 | 140/80 | 130/70 | 140/90 | 130/90 | | | 82 | 84 | 92 | 88 | 86 | 84 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 88 | 933090 | 24 | 98.8 | NO | NO | 130/80 | 130/80 | 140/80 | 140/70 | 140/90 | 130/80 | | | 84 | 86 | 84 | 86 | 86 | 88 | | | 24 | 24 | 24 | 24 | 24 | 24 | | | NO |
| 89 | 929328 | 22 | 98.6 | NO | NO | 130/80 | 110/70 | 140/80 | 130/80 | 140/90 | 140/80 | | | 82 | 86 | 80 | 82 | 84 | 88 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 90 | 927480 | 20 | 98.8 | NO | NO | 130/80 | 130/80 | 130/80 | 140/80 | 140/90 | 140/80 | | | 86 | 84 | 92 | 90 | 84 | 90 | | | 20 | 20 | 20 | 20 | 20 | 20 | | | NO |
| 91 | 929328 | 22 | 98.4 | NO | NO | 130/80 | 120/80 | 130/80 | 130/90 | 140/90 | 140/80 | | | 82 | 86 | 80 | 82 | 84 | 88 | | | 22 | 22 | 22 | 22 | 22 | 22 | | | NO |
| 92 | 944313 | 22 | 98.7 | NO | NO | 130/70 | 130/80 | 140/80 | 130/80 | 150/90 | 140/80 | | | 72 | 78 | 76 | 80 | 82 | 84 | | | 24 | 24 | 24 | 24 | 24 | 24 | | | NO |
| 93 | 943817 | 20 | 98.6 | NO | NO | 130/70 | 110/70 | 140/80 | 140/80 | 140/90 | 140/80 | | | 84 | 86 | 80 | 78 | 84 | 82 | | | 20 | 20 | 20 | 20 | 20 | 20 | | | NO |
| 94 | 934080 | 22 | 98.8 | NO | NO | 130/70 | 130/80 | 130/80 | 140/80 | 140/90 | 140/80 | 140/80 | | 88 | 84 | 90 | 92 | 96 | 94 | 92 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 95 | 678459 | 24 | 98.6 | YES | YES | 130/80 | 130/80 | 140/80 | 140/90 | 150/90 | 160/110 | | | 86 | 84 | 82 | 80 | 88 | 86 | | | 24 | 24 | 24 | 24 | 24 | 24 | | | YES |
| 96 | 945451 | 22 | 98 | NO | NO | 130/80 | 130/90 | 140/80 | 140/70 | 140/90 | 140/90 | 140/80 | | 84 | 80 | 86 | 90 | 94 | 98 | 96 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 97 | 813732 | 20 | 97 | NO | NO | 130/70 | 110/70 | 140/80 | 140/70 | 140/90 | 130/90 | 140/80 | | 82 | 88 | 86 | 90 | 94 | 92 | 96 | | 20 | 20 | 20 | 20 | 20 | 20 | 20 | | NO |
| 98 | 882380 | 22 | 98.8 | NO | NO | 130/70 | 130/90 | 140/80 | 140/90 | 140/80 | 140/80 | 130/80 | | 90 | 92 | 94 | 96 | 98 | 92 | 96 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |
| 99 | 303888 | 20 | 97 | NO | NO | 130/70 | 130/80 | 140/80 | 140/80 | 150/90 | 130/80 | 140/80 | | 86 | 84 | 82 | 80 | 86 | 88 | 92 | | 24 | 24 | 24 | 24 | 24 | 24 | 24 | | NO |
| 100 | 894637 | 24 | 99 | NO | NO | 130/70 | 120/80 | 140/80 | 140/90 | 140/90 | 140/70 | 140/80 | | 96 | 92 | 94 | 90 | 94 | 92 | 90 | | 22 | 22 | 22 | 22 | 22 | 22 | 22 | | NO |

| Sr. No | MRD NUMBER | Blood Group | Hemogram | Viral Markers | VDRL | FBS | TSH | Urine Routine | NST | IN HYPERTENSIVE PATIENTS_RFT | LFT | Uric Acid | LDH | Serum Proteins | Spot Protein: Creatinine Ratio |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | 791980 | O+ | HB,TLC,PLT-11.7,9.4,150 | NR | NR | 84 | 2.6 | ABNORMAL | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 6 | 0.2 |
| 68 | 891080 | B- | HB,TLC,PLT-10.3,12.5,229 | NR | NR | 88 | 1.72 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | ABNORMA | 6.4 | 0.3 |
| 69 | 890631 | B+ | HB,TLC,PLT-11.7,11.6,290 | NR | NR | 90 | 3.7 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.2 |
| 70 | 888167 | AB+ | HB,TLC,PLT-9.2,11.2,252 | NR | NR | 82 | 1.04 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6 | 0.3 |
| 71 | 894138 | B+ | HB,TLC,PLT-12.4,7.5,190 | NR | NR | 92 | 1.34 | NAD | REACTIVE | NORMAL | ABNORMAL | ABNORMA | NORMAL | 6.3 | 0.2 |
| 72 | 891992 | A+ | HB,TLC,PLT-10.8,10.2,219 | NR | NR | 94 | 1.8 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.5 | 0.3 |
| 73 | 558647 | B+ | HB,TLC,PLT-11.2,7.8,250 | NR | NR | 96 | 2.8 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.2 | 0.2 |
| 74 | 117378 | AB+ | HB,TLC,PLT-12.2,10.6,282 | NR | NR | 88 | 2.8 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 5.8 | 0.3 |
| 75 | 993566 | B+ | HB,TLC,PLT-13.2,14.8,155 | NR | NR | 96 | 2.9 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 76 | 893508 | B+ | HB,TLC,PLT-10.7,9.4,199 | NR | NR | 98 | 4.08 | ABNORMAL | REACTIVE | ABNORMAL | NORMAL | ABNORMA | ABNORMA | 6.3 | 0.2 |
| 77 | 28511 | B+ | HB,TLC,PLT-8,13.8,265 | NR | NR | 82 | 1.31 | NAD | REACTIVE | NORMAL | ABNORMAL | NORMAL | ABNORMA | 6.5 | 0.2 |
| 78 | 892668 | B+ | HB,TLC,PLT-12.7,10.5,248 | NR | NR | 96 | 2.8 | NAD | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 4.6 | 13.2 |
| 79 | 71774 | B+ | HB,TLC,PLT-14.7,13.3,173 | NR | NR | 84 | 2.4 | ABNORMAL | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 5.9 | 0.3 |
| 80 | 908477 | O+ | HB,TLC,PLT-14,11.5,347 | NR | NR | 88 | 4.8 | NAD | REACTIVE | ABNORMAL | ABNORMAL | ABNORMA | ABNORMA | 6.9 | 0.5 |
| 81 | 908501 | O- | HB,TLC,PLT-10,13.5,248 | NR | NR | 95 | 1.31 | ABNORMAL | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 7 | 0.3 |
| 82 | 908761 | O- | HB,TLC,PLT-13,11.4,100 | NR | NR | 89 | 2.4 | NAD | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 6.5 | 0.2 |
| 83 | 911028 | B+ | HB,TLC,PLT-9.5,9.6,262 | NR | NR | 115 | 3.42 | ABNORMAL | REACTIVE | NORMAL | ABNORMAL | ABNORMA | ABNORMA | 7.1 | 33.4 |
| 84 | 910716 | A+ | HB,TLC,PLT-12.2,11,178 | NR | NR | 100 | 2.8 | NAD | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 6 | 0.2 |
| 85 | 780034 | O+ | HB,TLC,PLT-14.1,10.8,256 | NR | NR | 92 | 2.4 | NAD | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 6.8 | 0.2 |
| 86 | 823208 | O+ | HB,TLC,PLT-8.9,11.6,183 | NR | NR | 105 | 1.77 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.7 | 0.2 |
| 87 | 878277 | B+ | HB,TLC,PLT-10,9.5,150 | NR | NR | 90 | 3.1 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 8 | 0.2 |
| 88 | 933090 | B+ | HB,TLC,PLT-11.9,10.9,254 | NR | NR | 98 | 3.7 | NAD | REACTIVE | NORMAL | NORMAL | ABNORMA | ABNORMA | 6.2 | 0.2 |
| 89 | 929328 | O+ | HB,TLC,PLT-11.6,11.6,150 | NR | NR | 88 | 3.9 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 90 | 927480 | B+ | HB,TLC,PLT-8.1,10.6,194 | NR | NR | 97 | 2.5 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.8 | 0.2 |
| 91 | 929328 | O+ | HB,TLC,PLT-11.6,11.1,150 | NR | NR | 88 | 3.9 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 92 | 944313 | O+ | HB,TLC,PLT-12.6,10.2,30 | NR | NR | 92 | 2.6 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.8 | 0.3 |
| 93 | 943817 | A+ | HB,TLC,PLT-9.9,10.2,152 | NR | NR | 98 | 1.17 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 94 | 934080 | A+ | HB,TLC,PLT-9.12,12.5,235 | NR | NR | 100 | 3.01 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.8 | 0.5 |
| 95 | 678459 | O+ | HB,TLC,PLT-7.3,11.6,101 | NR | NR | 88 | 2.2 | NAD | REACTIVE | ABNORMAL | ABNORMAL | NORMAL | NORMAL | 6.9 | 1.08 |
| 96 | 945451 | A+ | HB,TLC,PLT-8.4,11.2,240 | NR | NR | 90 | 4 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.6 | 0.4 |
| 97 | 813732 | B+ | HB,TLC,PLT-10.7,7.8,165 | NR | NR | 86 | 2.5 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.4 | 0.2 |
| 98 | 882380 | AB+ | HB,TLC,PLT-11.7,6.8,150 | NR | NR | 92 | 4.9 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.8 | 0.4 |
| 99 | 303888 | A+ | HB,TLC,PLT-10.9,11.7,159 | NR | NR | 100 | 3 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6 | 0.3 |
| 100 | 894637 | A+ | HB,TLC,PLT-10.4,12.3,199 | NR | NR | 90 | 2.5 | NAD | REACTIVE | NORMAL | NORMAL | NORMAL | NORMAL | 6.5 | 0.2 |

| Sr. No | MRD NUMBER | Fundus Examination | Dose of Antihypertensi | Route | Frequency | Duration (Months) | DEVELPOMENT OF HYPERTENSION | Date of birth | Time of birth | Sex | Weight | APGAR Score | NICU Admission | NEONATAL DEATH | comobidity |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | 791980 | MILD HTN RETINOPATHY | 100MG | P/O | BD | 3 | YES | 18/10/2019 | 4:02pm | F | 3.06 | 7 | No | No | PRETERM |
| 68 | 891080 | NORMAL | 200MG | P/O | TDS | 3 | YES | 20/10/2019 | 12:21pm | M | 2.62 | 6 | Yes | No | PRETERM |
| 69 | 890631 | NORMAL | 100MG | P/O | BD | 3 | YES | 23/10/2019 | 6:30am | F | 2040gm | 6 | Yes | No | - |
| 70 | 888167 | NORMAL | 200MG | P/O | BD | 3 | YES | 25/10/2019 | 11:40am | F | 3040gm | 9 | No | No | - |
| 71 | 894138 | NORMAL | 100MG | P/O | TDS | 4 | YES | 27/10/2019 | 4:40pm | F | 3.08 | 7 | No | No | - |
| 72 | 891992 | NORMAL | 200MG | P/O | BD | 4 | YES | 30/10/2019 | 9:55am | M | 2.66 | 9 | No | No | - |
| 73 | 558647 | NORMAL | 100MG | P/O | TDS | 3 | YES | 2/11/2019 | 10:48am | M | 3.3 | 10 | No | No | - |
| 74 | 117378 | MILD HTN RETINOPATHY | 100MG | P/O | BD | 4 | YES | 4/11/2019 | 10:18am | F | 3.09 | 9 | No | No | - |
| 75 | 993566 | NORMAL | 200MG | P/O | TDS | 3 | YES | 8/11/2019 | 6:18am | M | 3.5 | 7 | Yes | No | PRETERM |
| 76 | 893508 | MILD HTN RETINOPATHY | 100MG | P/O | BD | 3 | YES | 9/11/2019 | 8:32am | F | 1.04 | 4 | Yes | No | PRETERM |
| 77 | 28511 | NORMAL | 200MG | P/O | TDS | 3 | YES | 10/11/2019 | 3:53pm | M | 3.1 | 8 | Yes | No | PRETERM |
| 78 | 892668 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 3 | YES | 12/11/2019 | 8:38pm | F | 1.86 | 4 | Yes | No | PRETERM |
| 79 | 71774 | NORMAL | 100MG | P/O | TDS | 4 | YES | 13/11/2019 | 11:08am | M | 2.11 | 5 | Yes | No | PRETERM |
| 80 | 908477 | NORMAL | 200MG | P/O | TDS | 1 | YES | 15/11/2019 | 10:32am | F | 505gm | 4 | Yes | Yes | - |
| 81 | 908501 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 2 | YES | 17/11/2019 | 2kg | F | 3.3 | 7 | No | No | PRETERM |
| 82 | 908761 | NORMAL | 200MG | P/O | TDS | 4 | YES | 19/11/2019 | 11:24am | M | 2520gm | 7 | Yes | No | - |
| 83 | 911028 | MILD HTN RETINOPATHY | 200MG | P/O | TDS | 3 | YES | 20/11/2019 | 1:47am | F | 1880gm | 5 | Yes | No | PRETERM |
| 84 | 910716 | NORMAL | 100MG | P/O | BD | 1 | YES | 21/11/2019 | 5:51pm | M | 1300gm | 4 | Yes | No | PRETERM |
| 85 | 780034 | NORMAL | 200MG | P/O | TDS | 3 | YES | 24/11/2019 | 8:30am | F | 2900gm | 7 | Yes | No | - |
| 86 | 823208 | NORMAL | 100MG | P/O | BD | 3 | YES | 26/11/2019 | 1:44pm | M | 3440gm | 9 | No | No | - |
| 87 | 878277 | NORMAL | 200MG | P/O | BD | 3 | YES | 27/11/2019 | 2:44pm | F | 2160gm | 10 | Yes | No | - |
| 88 | 933090 | NORMAL | 100MG | P/O | TDS | 3 | YES | 29/11/2019 | 12:08pm | M | 1180gm | 4 | Yes | No | PRETERM |
| 89 | 929328 | NORMAL | 100MG | P/O | BD | 3 | YES | 3/11/2019 | 9:44am | M | 3060gm | 8 | No | No | - |
| 90 | 927480 | NORMAL | 100MG | P/O | TDS | 3 | YES | 5/12/2019 | 10:13am | F | 1880gm | 4 | Yes | No | - |
| 91 | 929328 | NORMAL | 100MG | P/O | BD | 3 | YES | 7/12/2019 | 9:44am | M | 3060gm | 8 | No | No | - |
| 92 | 944313 | NORMAL | 100MG | P/O | BD | 2 | YES | 8/12/2019 | 10:20pm | M | 2.8 | 7 | Yes | No | MAS |
| 93 | 943817 | NORMAL | 200MG | P/O | BD | 2 | YES | 12/12/2019 | 1:40pm | M | 3 | 9 | No | No | - |
| 94 | 934080 | NORMAL | 300MG | P/O | BD | 3 | YES | 18/12/2019 | 10am | F | 3.6 | 10 | No | No | - |
| 95 | 678459 | NORMAL | 100MG | P/O | TDS | 3 | YES | 19/12/2019 | 9:30am | M | 1455gm | 4 | Yes | Yes | PRETERM |
| 96 | 945451 | NORMAL | 200MG | P/O | BD | 2 | YES | 20/12/2019 | 11:08am | F | 2.3 | 6 | Yes | No | PRETERM |
| 97 | 813732 | NORMAL | 100MG | P/O | TDS | 3 | YES | 22/12/2019 | 2:16pm | M | 2540gm | 6 | Yes | No | MAS |
| 98 | 882380 | NORMAL | 200MG | P/O | BD | 2 | YES | 24/12/2019 | 10:55am | M | 2400gm | 6 | Yes | No | MAS |
| 99 | 303888 | NORMAL | 100MG | P/O | BD | 2 | YES | 26/12/2019 | 2:24am | M | 2802gm | 8 | No | No | - |
| 100 | 894637 | NORMAL | 100MG | P/O | BD | 4 | YES | 29/12/2019 | 9:25am | M | 3500gm | 9 | No | No | - |

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No. BFUHS/2K19p-TH/ 10407 Date 9/9/19

The Principal,
**DMC, Ludhiana.**

**Subject:** **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.05.2019 has approved the plan of thesis of **Anupriya Deep**, a student of your college for **MD Obstetrics & Gynaecology** along with the names of Supervisor/Co-Supervisor as under:-

**Topic Plan of Thesis**

**Location of placenta on ultrasound as a predictor test for hypertensive disease of pregnancy**

Supervisor
Dr. Sunil Kumar Juneja
Prof.,
Dept. of Obst. & Gynae.,
DMCH, Ludhiana.

Co-Supervisor
Dr. Ritu Dhawan Galhotra
Assoc. Prof.,
Dept. of Radio-Diagnosis,
DMCH, Ludhiana.

Dr. Kanupriya Jain
Asst. Prof.,
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DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,( if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

*Note :*

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

# LOCATION OF PLACENTA ON ULTRASOUND AS A PREDICTOR TEST FOR HYPERTENSIVE DISEASE OF PREGNANCY

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

In

OBSTETRICS & GYNAECOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

NOVEMBER, 2018

DR. ANUPRIYA DEEP

DEPARTMENT OF OBSTETRICS & GYNAECOLOGY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

# ABSTRACT OF PLAN OF THESIS

| | |
|---|---|
| Title | LOCATION OF PLACENTA ON ULTRASOUND AS A PREDICTOR TEST FOR HYPERTENSIVE DISEASE OF PREGNANCY |
| For the degree of | MD (OBSTETRICS & GYNAECOLOGY) |
| Name of the candidate | Dr. Anupriya Deep |
| Supervisor | Dr. Sunil Kumar Juneja, Professor, Department of Obstetrics & Gynaecology, DMCH, Ludhiana |
| Co-supervisor(s) | Dr. Ritu Dhawan Galhotra, Assoc Prof, Department of Radiology, DMCH, Ludhiana<br>Dr. Kanupriya Jain, Assistant Professor, Department of Obstetrics & Gynaecology, DMCH, Ludhiana |
| Institution | Dayanand Medical College and Hospital, Ludhiana |
| University | Baba Farid University of Health Sciences, Faridkot |

**Introduction**: Hypertensive disease of pregnancy, also known as maternal hypertensive disorder, is a group of diseases that includes preeclampsia, eclampsia, gestational hypertension, and chronic hypertension.

**Aims & Objectives**: To study location of placenta on ultrasound as a predictor test for hypertensive disease of pregnancy

**Material & Methods:** This study will be prospective study conducted in the department of obstetrics and gynaecology, Dayanand medical college and hospital, Ludhiana.

**Clinical Significance**: To decrease the complication rate, mortality and morbitity occuring due to hypertensive disease of pregnancy.

**Keywords:** Preeclampsia, eclampsia, gestational hypertension, and chronic hypertension.

| (Signature) Candidate | (Signature) Co-supervisor | (Signature) Co-supervisor | (Signature) Supervisor | (Signature) Principal |
|---|---|---|---|---|

## CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"LOCATION OF PLACENTA ON ULTRASOUND AS A PREDICTOR TEST FOR HYPERTENSIVE DISEASE OF PREGNANCY"** exist in the Department of Gynaecology, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

**Supervisor**

**Dr. Sunil Kumar Juneja**
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DMCH, Ludhiana

**Co-Supervisor(s)**

**Dr. Ritu Dhawan Galhotra**
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**Head of Department**

**Dr. Ashima Taneja**
Professor & Head
Department of Obstetrics & Gynaecology
DMCH, Ludhiana

Place : DMCH Ludhiana

Date : 15/12/18

# APPROVAL PROFORMA
# BY
# RESEARCH & ETHICAL COMMITTEE
# DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

| | |
|---|---|
| **Name of candidate** | Dr. Anupriya Deep |
| **Department** | Obstetrics & Gynaecology |
| **Topic of Thesis** | LOCATION OF PLACENTA ON ULTRASOUND AS A PREDICTOR TEST FOR HYPERTENSIVE DISEASE OF PREGNANCY |
| **Likely date of appearing in PG examination** | May 2021 |
| **Date of enrollment** | 9th May, 2018 |
| **Name of Head of Department** | Dr. Ashima Taneja |
| **1) Supervisor**<br>**2) Co-Supervisor(s)** | Dr. Sunil Kumar Juneja<br>Dr. Ritu Dhawan Galhotra<br>Dr. Kanupriya Jain |
| **Signature of Members of Research Committee with Stamp** | **Signature of Members of Ethics Committee with Stamp** |
| 1. HOD Pharmacology | 1. HOD Physiology |
| 2. HOD Surgery | 2. HOD Pathology |
| 3. HOD Pathology | 3. HOD Pharmacology |
| 4. HOD Medicine | 4. HOD SPM |
| 5. Dean Academics | 5. HOD Medicine |
| 6. Vice Principal | 6. HOD Surgery |
| | 7. HOD Obst. & Gynae. |
| | 8. Legal Expert |
| | 9. Secretary, IEC |
| | 10. Dean Academics |
| | 11. Vice Principal |
| | 12. Principal |

**Approved: Yes / No**

**Chairperson**
**Research Committee**

Chairperson
Research Committee

**Approved: Yes / ~~No~~**

**Chairperson**
**Ethics Committee**

Chairperson
Ethics Committee

# BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

## PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

| | | |
|---|---|---|
| 1 | NAME OF THE CANDIDATE AND ADDRESS | Dr. Anupriya Deep<br>Mehak hospital, Behrampur road, Gurdaspur, Punjab |
| 2 | NAME OF THE INSTITUTE | Dayanand Medical College and Hospital, Ludhiana |
| 3 | COURSE OF STUDY AND SUBJECT | MD, Obstetrics & Gynaecology |
| 4 | DATE OF ADMISSION OF COURSE | 9 May, 2018 |
| 5 | TITLE OF THE TOPIC | LOCATION OF PLACENTA ON ULTRASOUND AS A PREDICTOR TEST FOR HYPERTENSIVE DISEASE OF PREGNANCY |

## 6. BRIEF RESUME OF THE INTENDED WORK

### 6.1 NEED FOR THE STUDY

Pregnancy is one of the most cherished dreams in a woman's life. If there is any complication during pregnancy it may affect her mental and physical health. It is always good for the mother and the baby to detect any high risk factor so that it can be managed in time.

Hypertensive disease of pregnancy, also known as maternal hypertensive disorder, is a group of diseases which include preeclampsia, eclampsia, gestational hypertension, and chronic hypertension.

Preeclampsia is a complex clinical syndrome involving multiple organ systems and remains the one of the major cause of maternal and perinatal morbidity and mortality.[1] Women with a history of preclampsia have a high risk of development of preclampsia in subsequent pregnancies.

Hypertensive disorders are the most common medical condition complicating pregnancy. It complicates up to 7-10% of pregnancies of which Pre-eclampsia/ Eclampsia constitutes 70%, and Chronic hypertension 30%.[1] It is said that pre-eclampsia, eclampsia contribute to death of a woman every 3 minutes worldwide.[2] This has led to the interest in screening, in preventing this disorder. The most important factor is lack of timely prediction.[3]

Preclampsia is defined as persistent systolic blood pressure level of 140mm of Hg or higher or a diastolic blood pressure of 90mm of Hg or higher that occurs after 20 weeks of gestation with proteinuria in women with previously normal blood pressure.[4,5]

The incidence of preclampsia has increased by 25% during the past two decades.[6] Preclampsia is one of the leading cause of maternal and perinatal

morbidity and mortality with an estimated 50,000- 60,000 deaths per year worldwide.[7,8] For every preeclampsia related deaths that occur there are probably 50-100 other women who experience "near miss" significant maternal mortality that stops short of death but still results in significant health risk and health care cost.[9,10]

Those infants who are born small and premature may experience prolonged stay in neonatal intensive care units and often face developmental delays.

**MATERNAL COMPLICATION:-**

Outcome of pregnancy which is complicated by hypertension vary from uneventful pregnancy in women with chronic, controlled hypertension to death in cases of preclampsia-eclampsia. The major adverse outcome include central nervous system (CNS) injuries such as seizures , hemorrhagic and ischaemic strokes, hepatic damage ranging from transaminase elevation,"HELLP syndrome" (hemolysis, elevated liver enzymes and low platelets), hepatic failure, renal dysfunction (from trivial reduction in glomerular filtration to reversible acute liver failure or acute tubular necrosis to even irreversible renal failure secondary to renal cortical necrosis). There is also increased risk for women of preeclampsia to develop end stage renal disease though the absolute risk appears to be low. There is also increased frequency of cesarean delivery, preterm delivery and abruptio placentae.[11-13]

**FETAL COMPLICATION:-**

Preclampsia-eclampsia can lead to increased incidence of induced labour, fetal growth restriction, neonatal respiratory difficulties and increased frequencies of admission to neonatal intensive care units. The effect of chronic controlled hypertension in pregnancy on fetus are minimal.[11,13]

The significance of normal placentation for this cytotrophoblastic invasion is high and the cytotrophoblasts fail to adopt a vascular adhesion phenotype in preeclampsia. This may explain the reduced trophoblastic invasion in laterally situated placenta when the uteroplacental blood flow needs are mainly met by one side uterine artery.[14]

The overall risk of preclampsia in laterally located placenta is greatly increased due to inadequate development of blood supply.

Placenta plays one of the major role in the pathogenesis of preeclampsia. The blood supply of placenta is not uniformly distributed. As such, the site of implantation and resultant location of placenta with in the uterus are likely important determinant of placental blood flow and therefore pregnancy success.

The location of placenta is classified as central when it is equally distributed between right and left part of uterus irrespective of anterior, posterior, fundal. When 75% or more of placental mass is to one side of midline, it is classified as unilateral right or left.[15]

All women will be followed throughout pregnancy for the development of signs and symptoms of preclampsia.

## 6.2 REVIEW OF LITERATURE

Hypertension disorders of pregnancy, including pre-eclampsia complicate up to 5-10% of all pregnancies worldwide, constituting one of the greatest causes of maternal and perinatal morbidity and mortality worldwide.[16] Hypertensive disorders of pregnancy along with haemorrhage and infection constitute the deadly triad, which contributes greatly to maternal and mortality.

Hypertension is defined as either a systolic BP of 140 mm Hg or greater, a diastolic BP of 90 mm Hg or greater, or both. Hypertension is considered mild until diastolic or systolic levels are more than 110 mm Hg and more than 160 mm Hg, respectively. It is recommended that a diagnosis of hypertension requires at least two determinations of BP readings at least 4 hours a part. Although all occasion especially when faced with severe hypertension, the diagnosis can be confirmed within a shorter interval (even minutes) to facilitate timely antihypertensive therapy.[16]

Earlier known as pregnancy induced hypertension, HYPERTENSIVE DISORDERS OF PREGNANCY covers a spectrum of conditions, of which pre-eclampsia poses the greatest potential risk and remains one of the most common causes of maternal death.

**CLASSIFICATION: -**

1. Gestational Hypertension:-

Sustained systolic blood pressure at or above 140 mm of Hg or at a diastolic blood pressure of 90 mm of Hg, or greater after 20 weeks in the absence of accompanying proteinuria.[16] Increase in BP must be present on at least two separate occasions, 4 hours or more apart in pregnancy and hypertension resolves by 12 weeks postpartum.

2. Chronic hypertension of any etiology:-

Chronic hypertension is defined as high Blood pressure of more than systolic 140 and diastolic of 90 mm of Hg present before the 20th week.[17]

3. Pre-eclampsia superimposed on chronic hypertension:-

Onset of features diagnostic of pre-eclampsia in women with chronic hypertension beyond 20 weeks of gestation. If new onset or worsening baseline hypertension is associated by new onset proteinuria, then diagnosis of superimposed pre-eclampsia is made. It tends to be more severe and often accompanied by FGR.[16]

4. Pre-eclampsia and Eclampsia syndrome:-

- Preeclampsia

Preeclampsia is a syndrome that chiefly includes the development of new-onset hypertension in the second half of pregnancy. Although often accompanied by new-onset proteinuria, preeclampsia can be associated with signs and symptoms, including visual disturbances, headaches, epigastric pain, and the rapid development of edema.[16]

Diagnostic criteria include the development of hypertension, defined as a persistent systolic blood pressure (BP) of 140 mm Hg or higher, or a diastolic BP of 90 mm Hg or higher after 20 weeks of gestation in a women with previously normal blood pressure.[5,6] The optimal measurement of BP is made with the patient comfortably seated, legs uncrossed, and the back and arm supported, so that the middle of the cuff on the upper arm is at the level of the right atrium (the midpoint of the sternum). The patient should be made to relax and not talk during the measurement procedure; ideally, 5 minutes should elapse before the first reading is taken. If elevated on initial

assessment, the BP measurement should be repeated after some time to attempt to eliminate spuriously elevated BP determinations.[18] The measurement of BP taken in the upper arm with the women in left lateral position will falsely be lower BP recordings because cuff will be above heart.

Placenta is the vital link between the mother and the fetus for metabolic exchange, endocrine and other body functions. The blood supply of placenta is not uniformly disturbed.

As such the site of implantation and resultant location of placenta within the uterus are likely important predictors of placental blood flow and therefore pregnancy outcome. The two uterine arteries have a significant number of branches and each branch supplies the corresponding side of uterus.

In patients with centrally located placenta both the uterine arteries demonstrate similar resistance.[19] The uteroplacental blood flow are met equally by both of the uterine arteries.

However when the placenta is located laterally, the uteroplacental blood flow are met with one of the uterine artery with some contribution form the other uterine artery via the collateral circulation. The degree of collateral circulation may not be the same in all the subjects and deficient contribution may facilitate the development of preeclampsia and IUGR.[20]

Thus placental location determined by USG between 18-24 weeks is a non-invasive, cost effective, safe predictor of hypertensive disease of pregnancy.

## 6.3 AIMS AND OBJECTIVES OF THE STUDY

To study the correlation of placental location on ultrasound and development of hypertensive disease of pregnancy.

## 7. MATERIALS AND METHODS

### 7.1 Source of Data

This study will be prospective study conducted in the Department of Obstetrics and Gynaecology, Dayanand medical college and hospital, Ludhiana, which is a tertiary care hospital. This study will be conducted on outdoor as well as indoor patients between 18-24 weeks of gestation.

### 7.2 METHOD OF COLLECTION OF DATA

Study population will include antenatal cases on indoor as well as outdoor patients.

A structured predesigned proforma will be used for the study. Detailed history including basic demographics such as age, weight, height. BP etc will be taken. Relevant antenatal history and routine investigations will be recorded. Relevant past history and family history will be taken. The results obtained will be followed up and evaluated.

### INCLUSION CRITERIA

- All pregenant patients between 18-24 weeks of gestation.
- Patients consenting to participate in the study.

### EXCLUSION CRITERIA-

Subjects with concurrent diseases like :-

- Autoimmmune diseases.
- Lupus anticoagulant
- Anticardiolipin antibody
- Renal diseases.
- Connective tissue diseases.
- Thyrotoxicosis

Uterine anomalies

Substance abuse

- Alcohol
- Drug addiction.
- Drug

**OUTCOME**

Correlation of placental location and development of hypertensive disease of pregnancy and it's evaluation in terms of maternal and fetal outcome at the end of pregnancy.

**STATISTICAL ANALYSIS**

Data will be described in terms of range; mean ± standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups will be done using ANOVA. For comparing categorical data, Chi square ($\chi 2$) test was performed and exact test will be used when the expected frequency is less than 5. A probability value ($p$ value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

**7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.**

No

**7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?**

Yes attached herewith

## LIST OF REFERENCES


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## PROFORMA

Sr. No

Name

AGE

Weight

Height

HUSBAND'S NAME

DATE OF ADMISSION

ADMISSION NUMBER

MRD NUMBER

RESIDENTIAL ADDRESS

EDUCATION

OCCUPATION

PARITY

BOOKED

UNBOOKED

MENSTRUAL HISTORY

LMP

EDOD

POG

OBSTETRIC HISTORY

LOCATION OF PLACENTA ON ULTRASOUND

CALCIFICATION OF PLACENTA ON ULTRASOUND

PERIOD OF GESTATION WHEN SHE DEVELOPED HYPERTENSION

PAST HISTORY

FAMILY HISTORY

**Signature of Candidate**

**Signature of Supervisor**

SOCIO-ECONOMIC HISTORY

NUTRITIONAL HISTORY

PERIOD OF GESTATION AT THE TIME OF DELIVERY

MODE OF DELIVERY

VAGINAL (WITH FORCEPS/WITHOUT FORCEPS CAESAREAN

**MATERNAL COMPLICATIONS**

VOMITTING HEADACHE BLURRING OF VISION

EPIGASTRIC PAIN

**GENERAL PHYSICAL EXAMINATION**

PULSE BP RESPIRATORY RATE

TEMPERATURE

EDEMA

- GENERALISED
- PEDAL

| Gestation (weeks) | 18 | 20 | 22 | 24 | 28 | 32 | 36 | 40 |
|---|---|---|---|---|---|---|---|---|
| Blood Pressure | | | | | | | | |
| Pulse | | | | | | | | |
| Respiratory Rate | | | | | | | | |

**Signature of Candidate** **Signature of Supervisor**

**INVESTIGATIONS**

Blood Group

Hemogram

Viral Markers

VDRL

FBS

TSH

Urine Routine

NST

**IN HYPERTENSIVE PATIENTS**

RFT

LFT

Uric Acid

LDH

Serum Proteins

Spot Protein: Creatinine Ratio

Fundus Examination

**DRUGS USED IF THERE IS DEVELOPMENT OF HYPERTENSION**

Date

Dose

Route

Frequency

Duration

**Signature of Candidate** **Signature of Supervisor**